A Dissertation on

EFFECT OF INTRACUFF ALKALINIZED LIGNOCAINE ON THE INCIDENCE OF POST OPERATIVE SORE THROAT AND COUGH

submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

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for the award of the degree

M.D. (BRANCH-X)

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THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

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MAY 2019

DECLARATION BY THE CANDIDATE

I, DR. NASREEN KAJA , solemnly declare that the dissertation, titled **"EFFECT OF INTRACUFF ALKALINIZED LIGNOCAINE ON THE INCIDENCE OF POST OPERATIVE SORE THROAT AND COUGH "** is a bonafide work done by me during the period of APRIL 2018 to SEPTEMBER 2018 at Government Stanley Medical College and Hospital, Chennai under the expert supervision of **Dr. DHANASEKARAN M.D., D.A.,** Professor, Department Of Anaesthesiology, Government Stanley medical college,Chennai.

This thesis is submitted to The Tamil Nadu Dr. M.G.R. Medical University in partial fulfillment of the rules and regulations for the M.D. degree examinations in Anaesthesiology to be held in may 2019

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DR. NASREEN KAJA is an original work done in the Department of Anaesthesiology, Government Stanley Medical College and Hospital, Chennai in partial fulfilment of regulations of The Tamilnadu Dr. M.G.R Medical University for the award of degree of M.D. (Anaesthesiology) branch X, under my supervision during the academic period2016-2019.

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CHAPTER 1

INTRODUCTION

Post operative sore throat is the most common complaint after endotracheal intubation and is seen in upto 90% of the patients. It maybe considered along with endotracheal tube emergence phenomenon with laryngeal edema and ischemia. Chemical and mechanical irritation of the tracheal mucosa influences the incidence of cough at emergence from general anesthesia, potentially leading to significant postoperative complications.

Sore throat during emergence in a lighter plane of anaesthesia can result in detrimental hemodynamic changes. These changes are particularly undesirable in patients undergoing neurosurgical and, ophthalmic or those who have an increased risk of adverse cardiovascular event. Both in-vivo and in-vitro studies have been conducted on endotracheal tube cuffs filled with lignocaine. Past studies using high doses of lignocaine are associated with complications in the event of rupture of cuff. Sodium bicarbonate is hence added to increase the diffusion of lignocaine low

doses of through the cuff thereby preventing complications like sore throat, restlessness, nausea, vomiting, dysphonia and hoarseness of voice.

So in this study we compared the effect of intracuff alkalinized lignocaine and air in minimizing the incidence of post operative sore throat and cough after endotracheal intubation in general anaesthesia.

CHAPTER 2

AIM OF THE STUDY

PRIMARY OUTCOME:

To compare the incidence of post operative sore throat after general anaesthesia while using air and alkalinized lignocaine as the cuff inflation media of the endotracheal tube.

SECONDARY OUTCOME:

To study the incidence of cough, vomiting, hoarseness of voice and aspiration after extubation while inflating the cuff of the endotracheal tube with alkalinized lignocaine.

CHAPTER 3

HISTORY

The development of techniques and instruments for intubation ranks among the major advances in the history of anaesthesia[1]. The first use of elective oral intubation was undertaken by Scottish surgeon , William Macewan[2].

In 1926, Arthur Guedel began a series of experiments that lead to the introduction of the cuffed tube. He fashioned cuffs from the rubber of dental dams, condoms, surgical gloves that were glued onto the outer wall of the tubes[3].He recommended that the cuff be positioned just below the vocal cords to seal the airway. Waters later recommended that cuffs be constructed of two layers of soft rubber cemented together. These detachable cuffs were first manufactured by Waters' children.

Guedel sought ways to show the safety and utility of the cuffed tube. He first filled the mouth of an anaesthetized and intubated patient with water and showed that the cuff sealed the airway. He reasoned that if the cuff prevented water from

entering the trachea of an intubated patient , it should also prevent an animal from drowning even if it were submerged under water[4].

CHAPTER 4

ENDOTRACHEAL INTUBATION

Tracheal tubes are designed to provide a secured channel through the upper airway. The distal end lies in the mid to lower part of trachea, whereas the proximal end lies outside the mouth or nose where it is connected to the anaesthesia circuit or other device[5].Tracheal tubes used in adult patients have a cuff near the distal end that is inflated to provide a seal against the tracheal wall to protect the lungs from pulmonary aspiration and to ensure that the tidal volume delivered ventilates the lungs, rather than escapes into the upper airway. Cuffs are normally inflated with air and have an inflation tube with a pilot balloon that indicates cuff inflation[6].

Use of small tracheal tubes reduces the incidence of sore throat and hoarseness of voice. Small tracheal tubes may cause less tissue pressure in larynx. Cuff inflation achieves a seal between the tracheal tube and the wall of the trachea. There should be no air leak at airway pressures required for positive pressure

ventilation and the lungs should be protected from aspiration. The tracheal tube must be long enough for the cuff to lie 2 cm beyond the vocal cords.

PHYSIOLOGICAL RESPONSE TO TRACHEAL INTUBATION: [7]

Direct laryngoscopy and passage of a tracheal tube are noxious stimuli that provoke adverse responses in the cardiovascular, respiratory and other physiological systems. The magnitude of the response are greater with increase in force and duration of laryngoscope. Hemodynamic changes during tracheal intubation are undesirable in patients in cardiac disease and can result in myocardial ischaemia but seem to cause little harm to most patients. Many techniques have been tried to attenuate the stress response to intubation but none is ideal.

The ASA recommends that the anaesthesiologists have a preformulated strategy for extubation and management of post extubation problems.

Hypoventilation (residual effect of anaesthetic drug and neuromuscular blockade)
Upper airway obstruction
Laryngospasm and bronchospasm
Coughing (wound disruption)
Impaired laryngeal competence and pulmonary aspiration
Hypertension, tachycardia, dysrhythmia, myocardial ischemia

Table 1. Complications at extubation.

Extubation may be perfomed at different depths of anaesthesia. Deep extubation is performed to avoid adverse reflexes caused by the presence of the tracheal tube and its removal at a price of higher risk of hypoventilation and upper airway obstruction. Straining which could disrupt the surgical repair, is less likely with deep extubation.

Coughing maybe particularly troublesome during light plane of anaesthseiaextubation and cannot be entirely prevented. The frequency of cough may be reduced when the volatile used is sevoflurane. Intravenous lignocaine and alfentanil can also reduce coughing, as can local anaesthetic in the tracheal tube cuff are applied to the airway.

CHAPTER 5

CUFF OF THE ENDOTRACHEAL TUBE

A cuffed tracheal tube has an inflatable sleeve near the patient end of the tube. When this cuff is inflated to the desired volume , it seals the space between the tube and tracheal wall. It has an inflation tube connecting pilot balloon to the cuff. The pilot balloon has a one way inflation valve. In tubes which do not have an inflation valve, cuff inflation is maintained by applying a clamp to the external inflation tube or by applying a plug to its free end[(8)] .

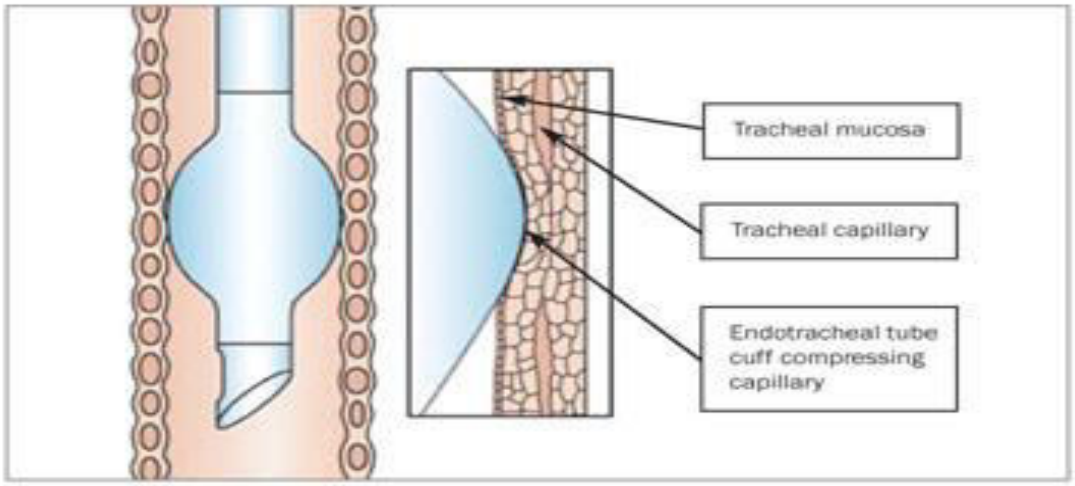


Fig 1:- Mechanism of tracheal mucosa perfusion injury secondary to ETT cuff overinflation.

Cuffs are classified, according to the pressure required to inflate the cuff, into low volume, high pressure cuffs and high volume, low pressure cuffs.

Low volume high pressure cuff – the red rubber tubes were made of a relatively low compliance thick rubber. These cuffs required a high pressure to distend them and were relatively low volume. These cuffs inflate in a circular shape rather than conforming to the shape of trachea. These cuffs have a small diameter at rest and a low residual volume. It has a small area of contact with the tracheal wall and distends and deforms the trachea in to circular shape. In order to achieve enough contact with the tracheal wall and a good seal, relative overinflation was required, with the result that the high pressure within the cuff was transmitted to the tracheal wall. This led to the increase in mucosal pressure to critical levels (capillary pressure is usually about 35 mmHg) could lead to mucosal ischemia, development of tracheal scarring and tracheal stenosis in case of prolonged surgeries.

Advantages of these cuffs include better protection against aspiration, better visibility during intubation than low pressure cuffs, lower incidence of sore throat.

High volume low pressure cuffs - They are made from thin inelastic material like PVC and h a large resting volume and diameter. As the cuff has a thin wall, it seals the trachea without stretching the tracheal wall. As the cuff is inflated, the area of contact becomes larger and the cuff adapts itself to the tracheal surface. The pressure within the cuff can therefore be kept much lower and can achieve a seal with minimal risk of occluding mucosal blood flow. The intracuff pressure closely approximates the pressure on the tracheal wall hence it is possible to measure and regulate the pressure exerted on the tracheal mucosa. Therefore there are less incidences of cuff related complications following prolonged intubation .

These cuff also have few disadvantages. It may be more difficult to insert as the cuff may obscure the view of the tip and larynx. A number of microfolds remain in the cuff even after a good seal and create microchannels running the length of the cuff. These channels may cause ventilator associated pneumonia by allowing the passage of infective pharyngeal contents beyond the cuff. When nitrous oxide is used, it will diffuse into the cuff causing an increased pressure on the tracheal mucosa.

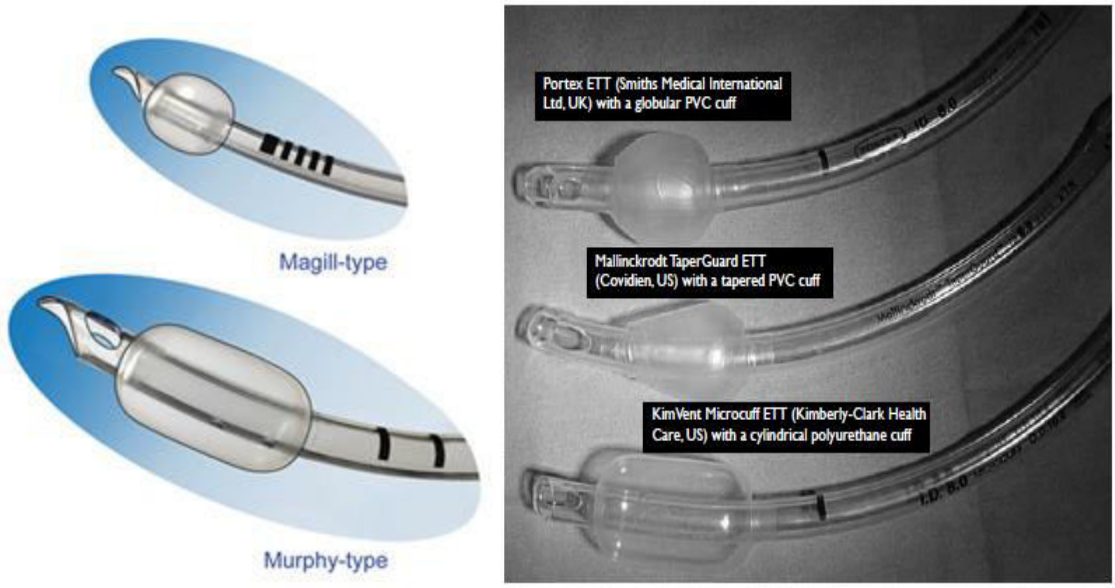


Fig 2:- Types of cuffs

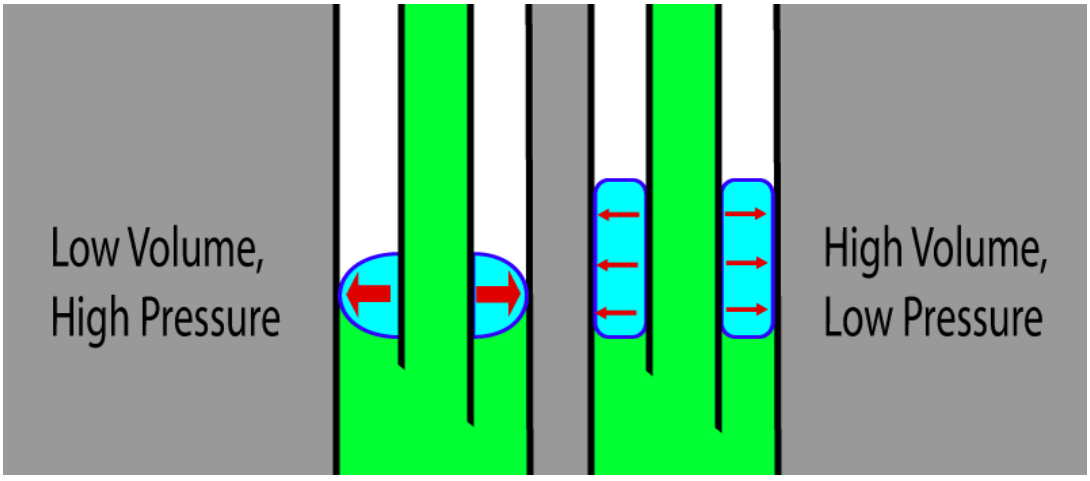


Fig 3:- Comparison of low volume high pressure and high volume low pressure cuffs

Foam cuff – It is made up of polyurethane foam and comes with bigger diameter and residual volume. The cuff is deflated by applying suction and when the negative pressure is released , the cuff expands. When in place, the amount that the foam expands determines the pressure exerted laterally on the tracheal wall and it is inversely proportional to each other. It provides a good seal at low tracheal wall pressure , when used according to appropriate size.

Fig 4:- Foam cuff

Lanz cuff - It comprises of a latex pilot balloon inside a transparent plastic sheath and has a pressure regulating valve between the cuff and the balloon. The pilot balloon is designed to maintain a intracuff pressure of 20 – 25 torr at the end of expiration. The pressure regulating valve prevents gas leak around the cuff during positive pressure ventilation and nitrous oxide related cuff pressure rise. How ever it may not give a good seal in patients requiring higher airway pressures.

Fig 5:-Lanz cuff

Fig 6:- ETT Cuff pressure monitoring

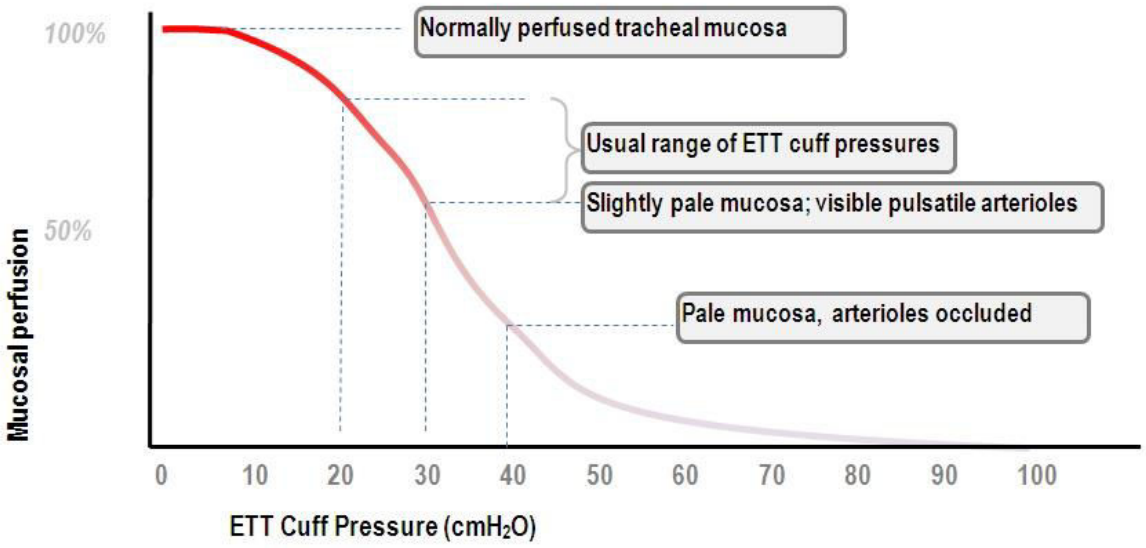


Fig 7:- Effect of ETT cuff pressure on tracheal mucosal perfusion

An optimal cuff pressure will give a good seal without compromising the blood supply of trachea. The recommended pressure on the lateral wall of the trachea is between 18- 25mmHg in normotensive adults[13].

Leak test is performed to measure the pressure exerted by the cuff of the tracheal tube on the tracheal mucosa. The valve on the breathing circuit is closed partially. The bag is squeezed with increasing pressure till an audible leak is

detected around the tube. The airway pressure at which the audible leak is detected is the pressure exerted by the tube on the tracheal mucosa.

The intracuff pressure and volume of a cuff inflated with air rise, when nitrous oxide is administered for a long time. The rate of diffusion depends on the permeability of the cuff material, the surface of the cuff exposed to nitrous oxide and partial pressure of nitrous oxide. When nitrous oxide administration is discontinued, the pressure in the cuff decreases rapidly. It can be measured by connecting the inflation tube to the pressure transducer of a monitor or direct measurement with a manometer.

CHAPTER 6

POST OPERATIVE SORE THROAT

Sore throat is a common postoperative complaint, occurring most often following endotracheal intubation. Main causative factors include tracheal-tube size and cuff design. Routine tracheal intubation for elective surgical procedures can result in pathological changes, trauma and nerve damage which may also account for postoperative throat symptoms. However, high intracuff pressure is associated with nerve palsies due to neuropraxia and nerve compression. Careful insertion techniques for the tracheal tube are of great importance in the prevention of airway trauma and postoperative sore throat.

Other factors that are found to be implicated were surgeries involving the head and neck region because of movement of the tube and cuff within the trachea and the presence of a nasogastric tube.

It has been clearly demonstrated that the use of a smaller tracheal tube reduces the incidence of sore throat, presumably because of decreased area of

contact between the tube and tracheal mucosa and hence decreased pressure at the tube–mucosal interface .

The tracheal-tube cuff has been implicated as a cause of serious sequelae following long-term intubation. The red-rubber tube had a low-residual-volume, high-pressure cuff and the exertion of this high pressure on the tracheal mucosa was thought to be damaging. A study of blood flow in rabbit tracheal mucosa demonstrated that when a high-pressure, low-volume cuff was inflated to >39cmH_2O , the mucosa in contact with the cuff, i.e. that covering the tracheal cartilage, became ischaemic[9]. This was thought to be due to a more even distribution of pressure over the mucosa. These low-volume high pressures cuffs still allowed some perfusion of the mucosa covering the cartilages[10]. When a thin-walled, low-pressure, high-volume cuff was used, blood flow did not cease until intracuff pressures was in the range 80–120 mmHg. Even so, the cautious recommendation was made that intracuff pressure should be maintained at <26cmH_2O.

After the introduction of high-volume, low-pressure cuffs, Loeser and co-workers[11,12,13,14,15] extensively investigated the effect of using tracheal tubes with different cuff designs on the incidence of postoperative sore throat, and showed that the high-volume cuffs were associated with a higher incidence of sore throat because of the greater area of cuff–tracheal contact. Although the high-volume cuffs caused a greater area of damage to the tracheal mucosa, the damage was more superficial than that caused by the high-pressure cuffs [16]. It is recommended that the ideal cuff should have a diameter slightly less than that of the trachea but should be constructed of material that would allow a 10% increase in diameter over the range of inflating pressure of 20–30 cmH_2O. In this way, wrinkling would be avoided, allowance could be made for variation in tracheal size when obtaining a seal and intracuff pressures would not compromise the tracheal mucosa. Furthermore, the cuff should be narrow in order to minimise the cuff–tracheal contact area[17].

High-volume, low-pressure cuffs will exert high pressure on the tracheal mucosa if overinflated following tracheal intubation, or if no allowance is made for N_2O diffusion. Both high- and low-volume cuffs undergo similar changes in

volume and pressure as a result of N_2O diffusion when inflated with air[18]. In an animal study, a comparison was made between cuff inflation with either saline or air. The pressure in the air-inflation group was significantly higher than that in the saline-inflation group [19].

The application of high pressure to the tracheal mucosa may also contribute to the occurrence of postoperative sore throat. The Brandt Anaesthesia Tube is designed to prevent intracuff pressure from increasing above 25cm H_2O, by virtue of the cuff communicating through the inflation line with a pilot balloon that is more compliant and of higher volume[20]. The incidence of postoperative sore throat in patients intubated with this tube (15%) is significantly lower than that after intubation with a standard Mallinckrodt tube (60%) which suggests that cuff-pressure limitation may help reduce the incidence of postoperative sore throat. Overinflation may further predispose the patient to postoperative sore throat by causing an increase in the cuff–tracheal contact area [21].

In order to avoid these problems, the cuff seal point should be carefully determined after tracheal intubation and that the intermittent measurement and adjustment of cuff pressure should be routine clinical practice [22]. Alternatively,

simple measures such as inflating the cuff with gas drawn from the breathing circuit or with saline will avoid the problem of N_2O diffusion.

Aneroid manometer
Brandt anesthesia tube system
VBM cuff pressure monitor
Tracoe cuff pressure gauge

Table 2:- Devices to control endotracheal tube cuff pressure

The effect of the application of laryngotracheal lignocaine spray on postoperative sore throat has also been investigated[23]. The incidence of sore throat was 29.2% in the study group and 19.6% in the control group. Although this difference was not statistically significant, it was concluded that the application of lignocaine spray could not be recommended for routine use. It was further

suggested that the lignocaine may be irritating or damaging to the tracheal mucosa[24].

There is no study therefore that categorically demonstrates that the use of lubricating jelly containing a local anaesthetic is beneficial in the reduction of postoperative sore throat after tracheal intubation. The application of lignocaine spray before intubation appears to increase the incidence of sore throat, as a result of either mucosal irritation or repeated laryngoscopy.

The role of suxamethonium in the aetiology of postoperative sore throat is unclear. It has been suggested that suxamethonium, which is known to cause postoperative skeletal muscle pain, could also lead to pain in the striated pharyngeal muscles, causing sore throat. In a study of 83 women undergoing dilatation and curettage who did not undergo tracheal intubation, the effect of administration of suxamethonium was examined[25]. Patients who received suxamethonium, either as a bolus or by infusion, had a significantly higher incidence of sore throat postoperatively. In summary, the use of smaller tracheal tubes with cuffs that have a small area of contact with the tracheal mucosa will reduce the incidence of postoperative sore throat. Careful control of intracuff

pressure may be beneficial even for short-term intubation, and consideration should be given to using either the anaesthetic gas mixture or saline to inflate the cuff. Lubricants containing local anaesthetic agents are not useful and may actually increase sore throat incidence

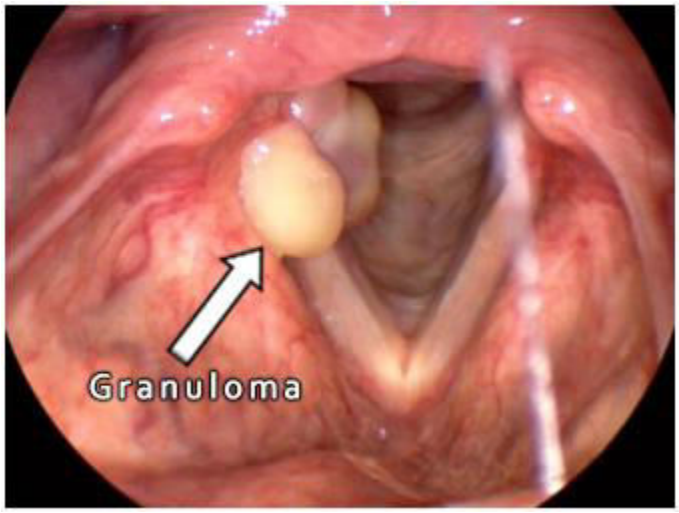
Pathological changes secondary to intubation include epithelial loss, glottis hematoma and edema, submucosal tears and contact ulcer granuloma.

Laryngeal trauma most commonly occurred posteriorly over the cricoid plate and also over the vocal processes of the arytenoids, as a result of forces exerted by the rigid tubes.

Traumatic injury to larynx during intubation can cause post operative sore throat because of neuropraxia due to high intracuff pressure and nerve demyelination due to gas sterilisation of the tubes. Decreased elasticity of the trachea and surrounding tissues in older people may also increase the likelihood of damage occurring during laryngoscopy and intubation.

Contact-ulcer granuloma is the most common late complication of tracheal intubation and should be suspected if the patient complains of prolonged

hoarseness. The site of the granuloma was usually at the tip of the vocal processes of the arytenoid cartilages, due to, among other things, their incessant movement[26].



` Fig 8:- Contact ulcer granuloma

Haematoma of the left vocal cord is the most common injury seen on indirect laryngoscopy which usually resolves spontaneously[27]. Optimisation of intubating conditions and careful technique are necessary to minimise airway trauma. Overinflation of the tracheal tube cuff has been associated with recurrent laryngeal nerve palsy and should be avoided. In summary, extensive damage to the

laryngeal and tracheal epithelia occurs as a result of tracheal intubation, even with an intubation period as short as 1hour.

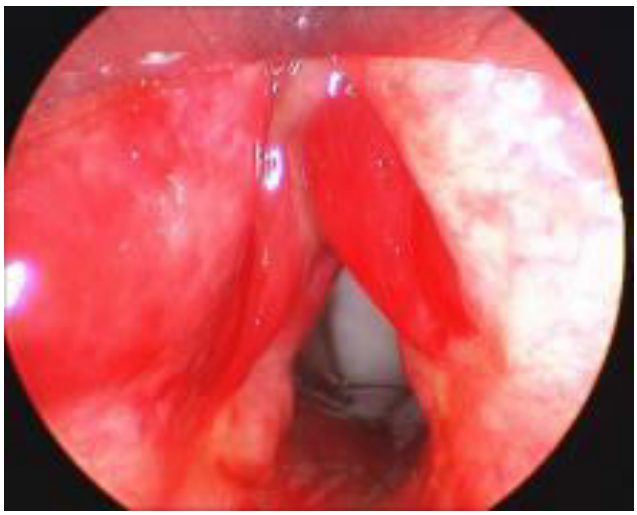


Fig 9:- Hematoma of vocal cord

In most cases, postoperative throat complaints resolve spontaneously without specific treatment. In moderate to severe cases it may be beneficial to treat pain and dysphagia with a gargle containing a drug such as benzydamine hydrochloride, which is approved for the symptomatic treatment of acute sore throat pain [28]. Benzydamine hydrochloride is a topical nonsteroidal anti-inflammatory agent that also has local anaesthetic activity.

Penetrating injuries caused by the laryngoscope blade, the airway or the patient's own teeth may require treatment with topical antibiotics [29].

Symptoms of postoperative throat discomfort such as sore throat, hoarseness and dysphagia are common, and are associated with trauma to the larynx and the pharynx. Careful airway management technique is therefore essential. Appropriate sizes of tracheal tube should be chosen. Lubricants containing local anaesthetics do not appear to be beneficial and may actually be harmful, having been implicated as a cause of bilateral recurrent laryngeal nerve palsy. Tracheal-tube cuffs that have minimal contact with the tracheal mucosa should be used, and monitoring and limitation of tracheal tube cuff pressures should be considered, both to reduce the incidence of postoperative sore throat and to minimise the risk of neuropraxia.

CHAPTER 7

LOCAL ANAESTHETICS

Local anaesthetics block voltage gated sodium channels and interrupt initiation and propogation of nerve impulses[30]. Currently available local anaesthtics belong to two chemical classes namely aminoesters and aminoamides.

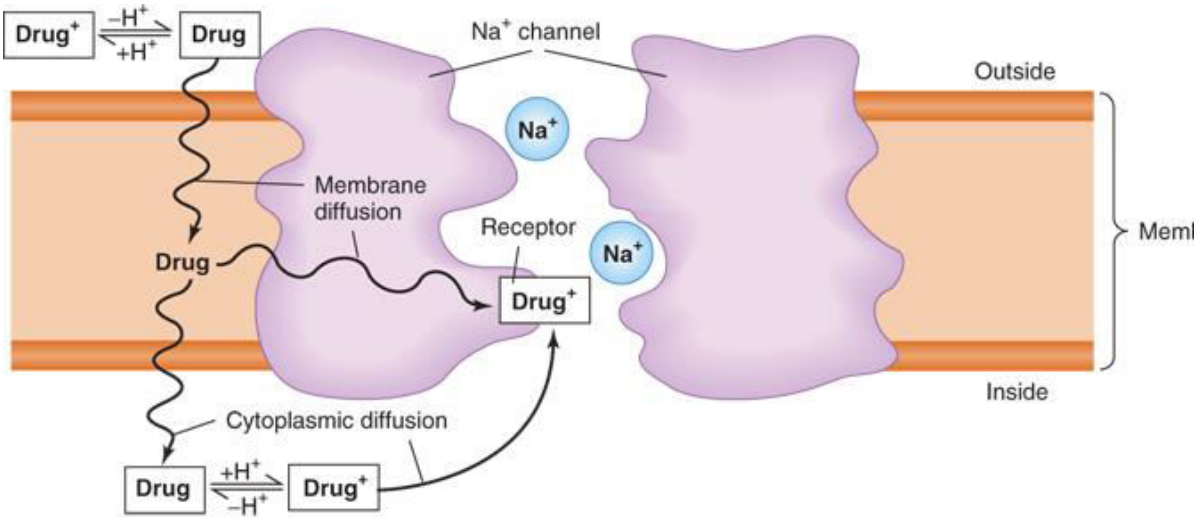


Fig 10:- Mechanism of action of local anaesthetics

Most of their features derive from the requirement for high solubility and rapid diffusion across biological membranes. Reversible protonation of the tertiary amine group tends to make local anaesthetic less charged at acidic pH, the neutral

base forms are more soluble in lipid environments. The typical local anaesthetic molecule contains a tertiary amine linked by an intermediate chain that always contains an ester or amide linkage. The aromatic hydrocarbon is hydrophobic in character whereas the tertiary amine is relatively hydrophilic. Local anaesthetic bases are poorly to sparingly soluble in water but are soluble in relatively hydrophobic solutions. Hence to optimize their shelf life most of the drugs are formulated as hydrochloride salts. Local anaesthetics with pKa nearest to physiological pH have the most rapid onset of action refelecting the presence of optimal ratio of ionized to non ionized fraction.

Aminoesters are metabolized primarily by plasma esterases and aminoamides by primarily enzymes. Recent efforts have led to the development of several new formulations of topical anaesthesia .

Hydrophobicity appears to be a primary determinant of the intrinsic anaesthetic potency. The onset of the conduction block in isolated nerves is related to physicochemical properties of the individual and the dose or concentration of the local anaesthetic used .

The duration of anaesthesia is markedly influenced by the peripheral vascular effects of the local anaesthetic, at low concentrations these agents tend to cause vasoconstriction , at higher concentrations they cause vasodilation. .

The addition of sodium bicarbonate to local anaesthetic solutions accelerates the time for achieving the required concentration(Cm) [30] needed for the conduction blockade and increases the amount of the uncharged base form, which should permeate the nerve sheath and nerve membrane. Hence sodium bicarbonate addition decrease the onset of action of the local anaesthetic solution.

In addition to blockade of impulses, local anaesthetic inhibits various receptors, and enhance the activity of certain intracellular signaling pathways.

Local anaesthetics in turn can suppress components of inflammatory responses by a cascade of systemic reactions.[31, 32]

Local anaesthetics are used to produce topical anaesthesia by placement on the mucous membrane of the nose, mouth, tracheobronchial tree, esophagus or genitourinary tract. Cocaine (4 – 10%), tetracaine (1-2%) and lignocaine (2-4%) are most often used topically. It is estimated that cocaine anaesthesia is used in

>50% of rhinolaryngeal procedures performed in the US [33].There is no difference between the intranasal anaesthetic or vasoconstrictive effect of cocaine and those of lignocaine – oxymetazoline mixture emphasizing its usefulness as a substitute for cocaine.

Nebulized lignocaine is used to produce surface anaesthesia of the upper and lower respiratory tract before fibreoptic bronchoscopy and as a treatment for patients experiencing intractable coughing [34].The inhalation of local anaesthetics by normal subjects does not alter airway resistance and may even cause bronchodilation. In contrast local inhalation of local anaesthetics can cause bronchoconstriction in some patients with asthma.

Local anaesthetics are absorbed into the systemic circulation after topical application to mucous membranes. Plasma lignocaine concentration 15 minutes after layngotrachealsparay are similar to the concentrations present at the same time after an IV injection of a similar dose of lignocaine. This reflects the high vascularity of the tracheobronchial tree and the injection of the local anaesthetic as a spray that spreads the solution over a wide surface area.

CHAPTER 8

LIGNOCAINE

In 1944, Nils Lofgren and Bengt Lundquist developed lignocaine – an amino amide local anaesthetic. It gained immediate popularity because of its potency, rapid onset and decreased incidence of allergic reactions and overall effectiveness for all types of regional anaesthetic blocks. Lignocaine contains a tertiary amine linked to an amide. The aromatic hydrocarbon is hydrophobic and lipophilic in character whereas the tertiary amine is relatively hydrophilic .The lipid solubility of lignocaine is 2.9.

Lidocaine alters signal conduction in neurons by prolonging the inactivation of the fast voltage-gated Na^+ channels in the neuronal cell membrane responsible for action potentialpropagation[30].

Potency	1
Onset	Rapid
Duration after infiltration	60-120 min
Maximum single dose for infiltration	300mg
Toxic plasma concentration	>5 micrograms/mL
pKa	7.9
Protein binding	70%
Fraction unionized at pH 7.4	25%
Lipid solubility	2.9
Volume of distribution	91L
Clearance	0.95 L/min
Elimination half time	96 min

Table 3 :- Pharmacology of lignocaine

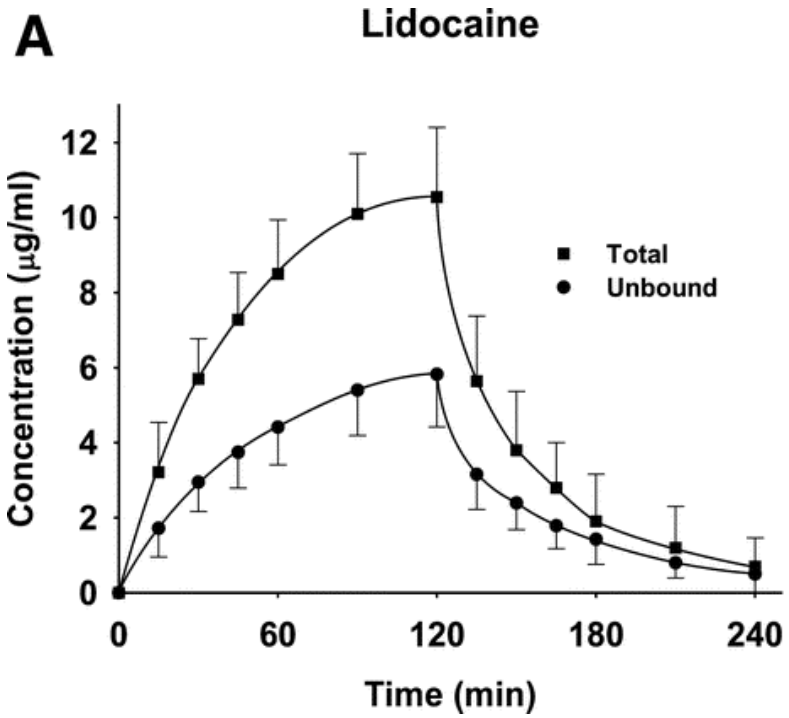


Fig 11:- Variation of plasma concentration of lignocaine with time

The principal metabolic pathway of lignocaine is oxidative dealkylation in the liver to monoethyglycinexylidide following by hydrolysis of this metabolite to xylidide. MEGX has approximately 80% of the activity of lignocaine for protecting against cardiac dysrhythmia in an animal model. This metabolite has a prolonged elimination half time accounting for efficacy of lignocaine infusion in controlling cardiac dysrhythmia. 75% of xylidide is excreted in the urine .

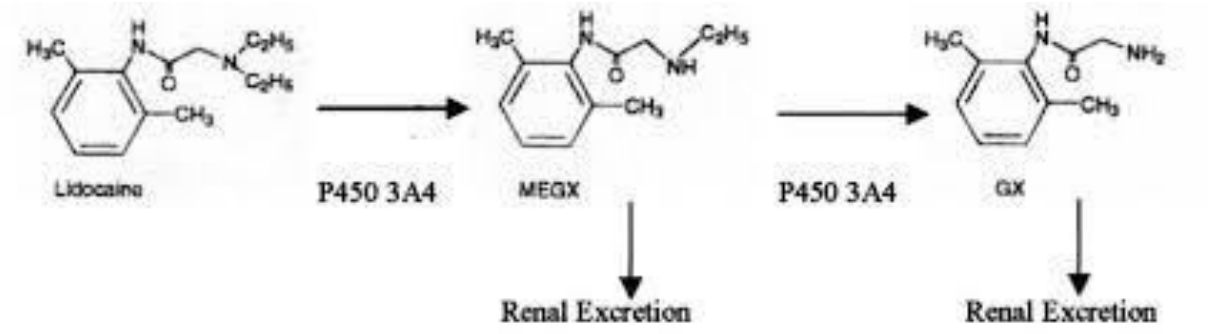


Fig 12:- Metabolism of lignocaine

Hepatic disease or decreases in hepatic blood flow which may occur during anaesthesia can decrease the rate of metabolism of lignocaine. Maternal clearance of lignocaine is prolonged in the presence of pregnancy induced hypertension and repeated administration of lignocaine can result in higher plasma concentrations than in normotensive pregnancy.

Plasma concentration of lignocaine (micrograms/mL)	**Effect**
1 – 5	Analgesia
5-10	Circumoral numbness
	Tinnitus
	Skeletal muscle wasting
	Systemic hypotension
	Myocardial depression
10-15	Seizures
	Unconsciousness
15-25	Apnea
	Coma
>25	Cardiovascular depression

Table 4:- Lignocaine toxicity

Clinical use	**Concentration (%)**	**Duration (min)**	**Maximum single dose (mg)**
Topical	4	30-60	300
Infiltration	0.5-1	60-240	300 or 500 with epinephrine
IVRA	0.25-0.5	30-60	300
Peripheral nerve block	1-1.5	60-180	300 or 500 with epinephrine
Epidural	1.5-2	60-120	300 or 500 with epinephrine
Spinal	1.5-5	30-60	100

Table 5:- Clinical usage of lignocaine

Lignocaine administered intravenously also decreases anaesthetic requirements for volatile drugs[35]. Lignocaine also may be administered

intravenously in the perioperative period as a cough suppressant . In this regard, the cough reflex during intubation of the trachea is suppressed by plasma concentrations of lignocaine > 2 micrograms /mL [36] .

Intravenous lignocaine has been postulated to decrease the post operative stress and improve anaesthetic depth. Lignocaine also possesses significant antibacterial effects[33]. Inhaled lignocaine attenuates histamine induced bronchospasm and induces airway anaesthesia reflecting topical airway anaesthesia.

CHAPTER 9

INTRACUFF ALKALINIZED LIGNOCAINE

Alkalinized lidocaine in the endotracheal tube (ETT) cuff decreases the incidence of cough and throat pain on emergence after surgery lasting more than 2 hours. However, alkalinized lidocaine needs 60–120 minutes to cross the ETT cuff membrane; therefore, its usefulness in shorter duration surgery is unknown.

During general anesthesia using nitrous oxide the cuff pressure increases as the temperature of the cuff rises and nitrous oxide diffuses into it more rapidly . This overinflation of the ETT cuff has been associated with damage to pharyngeal mucosa and recurrent laryngeal nerve palsy. These complications can be prevented by filling the ETT cuffs with lignocaine.

Alkalinization of lignocaine shortens the onset of a neural blockade, enhances the depth of blockade and increases the spread of the blockade. The pH of commercial preparation of local anesthetics range from 3.9 to 6.5. Alkalinization increases the percentage of local anaesthetic existing in the lipid soluble form that is available to diffuse lipid cellular barriers.

Lidocaine when used as endotracheal tube (ETT) cuff inflation media reduces the postintubation related sore throat and cough. A lower incidence of endotracheal tube discomfort and sore throat after 24 hours and lower systolic arterial pressure at the time of extubation has also been observed when lignocaine is used as the cuff inflation media [36]. Tracheostomized patients, who have to keep the tube for a long time and whose discomfort seems to come mainly from the inflated cuff, can benefit from intracuff lidocaine[37]. Pressures in cuffs filled with lignocaine were significantly lower than in cuffs filled air.

For patients with high peak inflation pressure, an injection of a large volume of lignocaine into the cuff is needed because the minimum occlusive pressure increases linearly with peak inflation pressure [38]. It should be ascertained, not to use more than the maximum allowable dose.

In addition, intracuff lidocaine does not increase the cuff volume during the maintenance of general anesthesia with nitrous oxide. It would be more effective for certain kinds of surgery like neuro or craniofacial surgery because this type of surgery usually takes long time, and the pilot balloon port cannot be accessible for the cuff pressure adjustment without interrupting the surgical procedure.

CHAPTER 10

REVIEW OF LITERATURE

1. Huang et al[39] reported that alkalization of lidocaine can promote the *in vitro* diffusion across the endotracheal tube cuff many tens of times. The alkalization of lidocaine and the surgery of long duration would make the method more effective. Use of a heated breathing circuit may have some additive effect because warming of lidocaine solution can increase the diffusion across the cuff membrane.

2. Loeser *et al*[40] in their found that the use of uncuffed tubes resulted in a significantly higher incidence of sore throat than the use of cuffed tubes, even when the patients breathed warmed and humidified gases. It was thought that this could be due to nonhumidified air being drawn across the airway mucosa during spontaneous respiration. There was a higher incidence of sore throat when all the cuffed tubes were lubricated with lignocaine ointments, as opposed to a water-soluble jelly or no lubricant at all. However, the incidence was as high as 90% when the uncuffed tubes were lubricated with 4% lignocaine jelly, and the

severity of sore throat in these patients was significantly greater. Conversely, a comparison between intubation with dry tubes or a tube lubricated with jelly containing 1% cinchocaine led the investigators to suggest that the use of lubricants containing a local anaesthetic may be beneficial. Of the 248 patients in that study, 39% who were intubated with a dry tube complained of sore throat on the first postoperative day compared with 24% who were intubated with a lubricated tube, a significant difference. After the first postoperative day, the incidence decreased rapidly in both groups. A further comparison was made in 60 patients between lubrication of the tube with jelly containing cinchocaine and lubrication with the same jelly without cinchocaine[40]. The incidence of sore throat was 38% in the non cinchocaine group vs 25% in the cinchocaine group, which was not statistically significant, although it might have become so with greater numbers of patients. Subgroup analysis revealed that both alkalinized lidocaine and non-alkalinized lidocaine offered protection compared with control groups. Regarding the pain intensity of post operative sore throat at 1 h, the mean in intervention group was 14.1 mm on Visual Analogue scale while the control was

29.1 mm; the mean difference between lidocaine and the control was significant both in the alkalinized and non-alkalinized subgroups.

3. Prerana P Shroff et al [41] conducted a prospective randomized, controlled study over 3 months and examined the efficacy of different media used for inflation of tracheal tube cuffs. The patients were divided into three equal groups (air, isotonic saline and alkalinized lignocaine as inflation media) using sealed envelope technique. The volume of the inflation medium, intracuff pressure, duration of intubation, volume of the inflation medium withdrawn from the cuff and complications like tube intolerance, coughing on tube, restlessness, hoarseness, sorethroat, breathlessness and laryngospasm were analysed. After intubation at all intervals, the intracuff pressure was higher in the air group with statistical significance at 5 min, 30 min, 1 h and just before extubation when air and saline groups were comparable and at all intervals after intubation up to just before extubation when air and lignocaine groups were comparable. The volume of air increased just before extubation in the air group, as compared with a fall in volume in the other groups. Tube intolerance, hoarseness and sore throat were least in the

lignocaine group. Hence, they concluded that alkalinized 2% lignocaine and saline are better cuff inflation media, than air.

4. PapuNath et al [(42)] conducted a prospective double-blind randomized controlled trial to test the hypothesis that alkalinized lidocaine would reduce the incidence of emergence cough after surgeries lasting <120 minutes. American Society of Anesthesiologists PS I–III patients were randomized into 1 of 2 groups receiving either alkalinized lidocaine (group AL) or saline (group S) to inflate the ETT cuff. Cuffs were prefilled >90 minutes before intubation with either 2 mL of 2% lidocaine and 8 mL of 8.4% bicarbonate (group AL) or 10 mL of normal saline (group S). Cuffs were emptied immediately before intubation. After intubation, either 2 mL of 2% lidocaine (AL) or 2 mL of saline (S) were injected into the cuff. Additional 8.4% bicarbonate (AL) or saline (S) was injected into the cuff until therewas no air leak. Anesthesia was maintained using desflurane, rocuronium, and either fentanyl or sufentanil to maintain vital signs within 20% of baseline values. Opioids administered in prophylaxis of extubation cough were proscribed. The incidence of extubation cough in group AL was 12%, significantly lower (1-sided

P = .045) than the 22% incidence in group S. The 1-tailed risk ratio for cough in group AL was 0.55 (0–0.94, P = .045). the conclusion derived was that alkalinized lidocaine in the ETT cuff significantly decreased general anesthesia emergence cough after surgeries with an average duration of slightly <1 hour.

5. P.do Nascimento et al [43] studied laryngotracheal morbidity in children after tracheal intubation using tracheal tube cuffs filled with alkalinised lidocaine. They used 20 cmH2O for the cuff pressures and the pressure transmitted to the tracheal mucosa against the capillaries was based on Pascal's principle. The formula 'p=ρgh' was applied. They concluded that there was decreased incidence of cough following extubation when alkalinized lignocaine was used to inflate the cuff.

6. In a study conducted by Ahmed et al [44] a total of 64 patients who expected to require ventilatory support for a period of more than 48 h were randomly assigned to groups S and L. In group S, the endotracheal tube (ETT) cuffs were inflated with normal saline. In group L, the ETT cuffs were inflated with lidocaine 2% and sodium bicarbonate 8.4%. The investigator and the surgical intensive care unit staff

were blinded to the nature of cuff-filled solutions. Sedation was maintained with propofol and fentanyl infusions. The total requirements for propofol and fentanyl, frequency and severity of cough and number of ineffective triggering during the first 24 h of mechanical ventilation were recorded.

There was a significant reduction (about 30%) in the requirements for propofol and fentanyl in patients who received intracuff alkalinized lidocaine; $P < 0.001$. The frequency and severity of cough were significantly lower in group L compared with group S and the frequency of ineffective triggering was significantly lower in group L; $P < 0.001$ for both comparisons. They concluded that Intracuff alkalized lidocaine increases ETT tolerance and hence, decreases sedatives/analgesics requirements for mechanically ventilated patients. This results in improved patient-ventilator synchronization.

7. Shroff PP, Patil V. [45] examined the efficacy of different media used for inflation of tracheal tube cuffs. In their prospective randomized, controlled study over 3 months, there were 150 patients of either sex undergoing surgery under general anaesthesia with controlled ventilation with nitrous oxide and oxygen. The

patients were divided into three equal groups (air, isotonic saline and alkalinized lignocaine as inflation media) using sealed envelope technique. The volume of the inflation medium, intracuff pressure, duration of intubation, volume of the inflation medium withdrawn from the cuff and complications like tube intolerance, coughing on tube, restlessness, hoarseness, sore throat, breathlessness and laryngospasm were analysed. The intracuff pressure was higher in the air group with statistical significance at 5 min, 30 min, 1 h and just before extubation when air and saline groups were comparable and at all intervals after intubation up to just before extubation when air and lignocaine groups were comparable. The volume of air increased just before extubation in the air group, as compared with a fall in volume in the other groups. Tube intolerance, hoarseness and sore throat were least in the lignocaine group. They found that alkalinized 2% lignocaine and saline are better cuff inflation media, than air.

8. In a study conducted by Yoshihiro Momota et al [(46)] lidocaine hydrochloride and alkalinized lidocaine hydrochloride solutions were filled in endotracheal tube cuffs to determine the rate of diffusion of lidocaine across the cuffs, and assess the usefulness of these cuffs as a drug delivery system.

Oral RAE® tracheal tubes were filled with three different lidocaine solutions, i.e., mixtures of 4% lidocaine hydrochloride solution and distilled water, 4% lidocaine hydrochloride solution and 8.4% sodium bicarbonate solution (LSB-Gr), and 4% lidocaine hydrochloride solution and dipotassium phosphate solution (LDP-Gr). Cuffs filled with the relevant lidocaine solution were placed in beakers filled with distilled water. A 100 μL sample of the water in the vessel was taken from each beaker every 30 minutes for 360 minutes to determine the concentration of lidocaine diffused across the cuff using fluorescence polarization immunoassay. The cuff surface was observed after 60, 180, and 360 minutes of exposure for changes in the structure of the material. The results indicate that alkalinization of intracuff lidocaine increases the rate of diffusion of lidocaine across the endotracheal tube cuff.

9. In a randomized controlled trial by Souissi et al[47], they concluded that Intracuff 160 mg alkalinized lidocaine reduces cough upon emergence from N2O-free general anesthesia. This study evaluated the benefits of endotracheal tube (ETT) intracuff alkalinized lidocaine during N2O-free general anesthesia by assessing the

in vitro effect of alkalinization on lidocaine diffusion kinetics across the cuff's membrane and evaluating, in a randomized controlled clinical trial, the impact of 160 mg of intracuff alkalinized lidocaine on cough upon emergence from anesthesia for surgery lasting > 120 min. In the clinical trial, 80 adult patients (American Society of Anesthesiologists physical status I-III) undergoing urological or gynecological surgery expected to last > 120 min and scheduled for N2O-free general anesthesia were enrolled. Their in vitro study confirmed that alkalinization increases lidocaine diffusion across the membrane of ETT cuffs and suggested that the lidocaine diffusion rate is associated with the initial intracuff lidocaine quantity. Their clinical trial demonstrated that, compared with the saline group, 160 mg of intracuff alkalinized lidocaine reduced the incidence of cough upon emergence from N2O-free general anesthesia (76% vs 34%, respectively; difference 42%; 95% confidence interval, 21% to 62%; $P < 0.001$) while having no clinical impact on secondary outcomes.

10. Vandse R et al[48] in a Randomized Double Blind Control study compared the efficacy of intracuff alkalinized lidocaine to low dose Remifentanil infusion in

attenuating the endotracheal tube induced emergence Phenomena.120 ASA I-III patients, aged 18-65 years, were randomly assigned to receive intracuff alkalinized lidocaine (2% lidocaine mixed 1:1 with 1.4% NaHCO3) or an intravenous remifentanil infusion (0.05-0.5 mcg/ kg/min) combined with intracuff saline during desflurane-based general anesthesia. At the end of surgery, after desflurane was turned off in the assigned group, low dose remifentanil, or its equivalent placebo was decreased to one-tenth of the mean dose but not less than 0.01 mcg/kg/min and it was continued until extubation. A blinded researcher observed each patient from the time desflurane was discontinued until at least five minutes after extubation. Coughing was evaluated as either present or not, and graded on a point scale based on severity. The patients were also observed for development of any adverse events along with the vital signs during this emergence phase.

The incidence (44% vs 67%, p=0.02) and severity of coughing, overall, was significantly less in the lidocaine group compared to remifentanil group. The lidocaine group also had a lower incidence of significant coughing (25% vs 49%, p=0.009). The mean arterial pressure (MAP) in the lidocaine group was lower than the remifentanil group at extubation and 5 minutes after extubation. It was

concluded thatIntracuff alkalinized lidocaine (ICL) is more effective in reducing the incidence and severity of coughing compared to a low dose remifentanil infusion during emergence from desflurane based anesthesia.

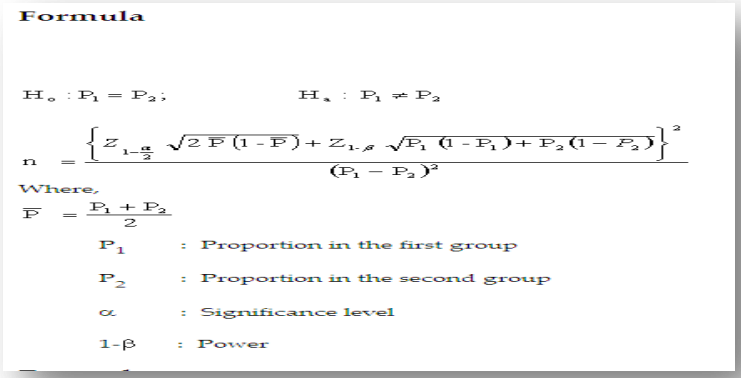
CHAPTER 11

MATERIALS AND METHODS

This study was done in patients undergoing elective surgical procedures lasting for more than two hours under general anesthesia in the Department of Otorhinolaryngology and Department of General Surgery, Stanley Medical college, Chennai. This was a randomized, prospective comparative study conducted on 150 patients over a period of six months. Patients were explained about the procedure in detail and informed written consent was obtained. The approval of the Institutional Ethical Committee was obtained.

SAMPLE SIZE AND RANDOMIZATION

The sample size of 75 was arrived with the formula given below:



Formula

$H_0 : P_1 = P_2 ; \qquad H_a : P_1 \neq P_2$

$$n = \frac{\left\{ Z_{1-\frac{\alpha}{2}} \sqrt{2\,\bar{P}\,(1-\bar{P})} + Z_{1-\beta} \sqrt{P_1\,(1-P_1) + P_2(1-P_2)} \right\}^2}{(P_1 - P_2)^2}$$

Where,

$$\bar{P} = \frac{P_1 + P_2}{2}$$

P_1 : Proportion in the first group

P_2 : Proportion in the second group

α : Significance level

$1-\beta$: Power

To find the significant difference between the bivariate samples in Paired groups the Paired sample t-test was used & for Independent groups the Unpaired sample t-test was used.

To find the association of significance in categorical data the Chi-Square test was used.

In all the above statistical tools the probability value 0.05 will be considered as significant level.

STUDY DESIGN

Prospective , randomized , comparative, single blinded study was conducted.

PRE ANAESTHETIC EVALUATION

Patients aged between 18-60 years scheduled to undergo elective surgical procedures lasting for more than 120 min under general anaesthesia were subjected to pre anaesthetic evaluation in our study.

Routine blood investigations were evaluated which included hemoglobin, hematocrit, blood urea, serum creatinine. Detailed history about past medical diseases like Diabetes mellitus, hypertension, Asthma, Tuberculosis, Seizure disorder, previous intake of medications and vices like smoking, tobacco chewing, alcohol consumption were elicited. History about previous surgery performed under general anesthesia was also elicited.

Airway assessment was made based on Modified Mallampati classification, range of neck movements and upper lip bite test.

Examination of cardiovascular, respiratory and neurological system was carried out in all patients included in the study .

Patients were assessed under the American Soceity of Anaesthsiologists – Physical Status classification.

Patients not fulfilling the inclusion criteria were excluded from the study.

INCLUSION CRITERIA

All patients assessed under ASA physical status classification I and II aged 18-60 yrs scheduled to undergo surgery under general anaesthesia lasting for more than 2 hours.

EXCLUSION CRITERIA

1. Patients with difficult airway i.e. Modified Mallampatti grade III/IV,
2. Patients with history suggestive of Gastro esophageal reflux,
3. Patients with history of laryngeal or tracheal surgery and history of asthma, cardiovascular disease, smoking.
4. Recent respiratory infection,

5. Requirement of more than one attempt for endotracheal intubation
6. Patients who need nasogastric tube intraoperatively
7. Surgery lasting less than 40 min
8. No consent for study.

MATERIALS

The equipments, drugs, and monitors required for endotracheal intubation were kept ready for the conduct of anaesthesia :

EQUIPMENTS :

1. Oxygen source
2. Suction apparatus
3. Cuff pressure manometer
4. End tidal carbon dioxide analyzer

MONITORS:

Continuous ECG, Pulse oximetery, Non invasive blood pressure, End tidal carbon dioxide analyzer.

METHODOLOGY

Subjects were allocated into two groups, Group(A) where the endotracheal tube cuff filled with alkalinized lignocaine 6.5 ml (130 mg) made alkalinized with 1.4% sodium bicarbonate 1.5 ml (the sodium bicarbonate 7.5% is available) and Group (B) were the endotracheal tube cuff is filled with intracuff air according to computer generated randomization sheet.

Standard monitors were attached which include ECG, NIBP, SPO2; ETCO2. Study subjects were premedicated with InjGlycopyrrolate 0.005mg/kg body weight, Inj Midazolam 1mg, Inj Fentanyl 2 microgram/kg body weight. Baseline hemodynamic parameters being noted. Patients were preoxygenated with 100% oxygen for 3 min. and induced with Inj. Thiopentone 5mg/kg body weight, Inj. Atracurium 0.5mg/kg .

Female and male patients were intubated with ET tube size of 7mm and 8.5mm respectively. The endotracheal tube was inflated to cuff pressure set at 20 cm of water . For group (B) the amount of air required to attain the set cuff pressure that is 20 cm of water measured and the same amount of alkalinized

lignocaine injected to inflate the cuff. Cuff pressure was recorded using a cuff pressure manometer at the start and end of surgery .

Anaesthesia maintained with Nitrous: Oxygen (35:65), InjAtracurium top up was administered, 1/4th the intubating dose, depending upon the EtCO2 changes. Lungs were mechanically ventilated with tidal volume of 8-10ml/kg body weight. Pulse oximeter, Non invasive blood pressure, ECG and EtCO2 monitored

At the end of surgery, Patient reversed with InjGlycopyrrolate 0.005 mg/kg body weight and Inj Neostigmine 0.05 mg/kg body weight. Cuff pressure was measured just before deflating the cuff and recorded. Extubation performed after checking for adequate spontaneous ventilation, ability to follow verbal commands (eye opening, tongue protrusion) , presence of protective airway reflexes and return of adequate muscle power.

Patients were assessed for sore throat, at 0 minutes, 1 hour, 6 hours, 12 hrs& 24 hours after extubation. Sore throat was recorded and graded according to the post operative scoring system. Other features like post extubation cough, nausea, vomiting, hoarseness of voice and evidence of aspiration were recorded as either

occurred or not occurred by another anaesthesiologist who was not present at the time of intubation. We recorded any adverse events intraoperatively and post operatively.

Grade	Severity
	Postoperative sore throat
0	No sore throat at any time since the operation
1	Minimal - Patient answered in the affirmative when asked about sore throat
2	Moderate - Patient complained of sore throat on his/her own
3	Severe - Patient is in obvious distress

Table 6:- Post operative sore throat grading [49]

CHAPTER 12

OBSERVATIONS AND RESULTS

FINDINGS:

A prospective observational study on the effect of intracuff alkalinized lignocaine on the incidence of post operative sore throat and cough after general anaesthesia in a tertiary care center revealed the following findings. All patients assessed under ASA physical status classification I and II aged 18- 60 yrs scheduled to undergo surgery lasting for more than 2 hours were included in the study.

Age distribution

The following figures 1 and 2 shows the age distribution of the patients in the air group and lignocaine group respectively. The mean age group (in years) in air group is 32.96 (S.D=10.44) and in lignocaine group is 40.08 (S.D=13.928).

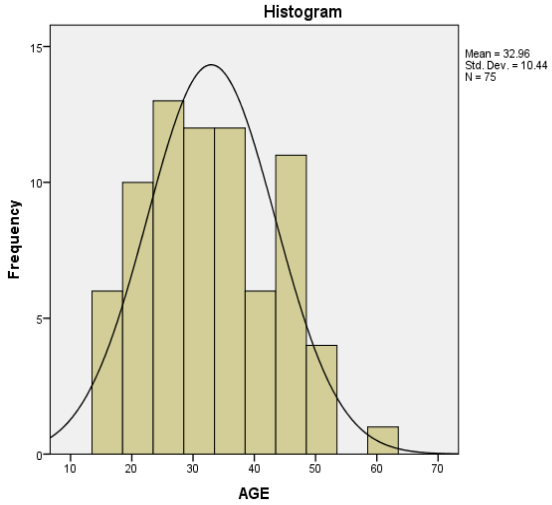


Figure 13:- Age distribution in air group

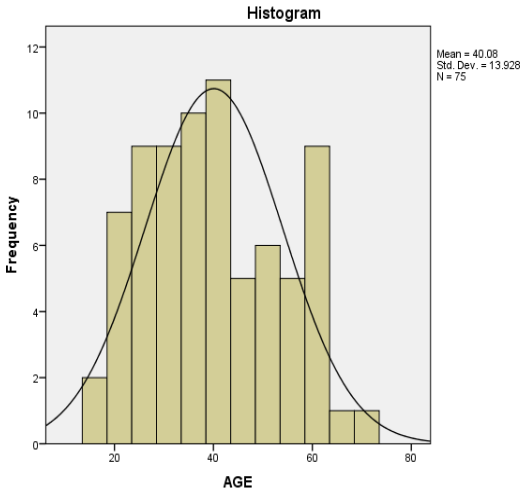


Figure 14:- Age distribution in lignocaine group

Gender distribution among the samples

The following figure and table shows the gender distribution among the samples. Females were more in number in each of the groups (Air=54.7%;Lignocaine=50.7%).

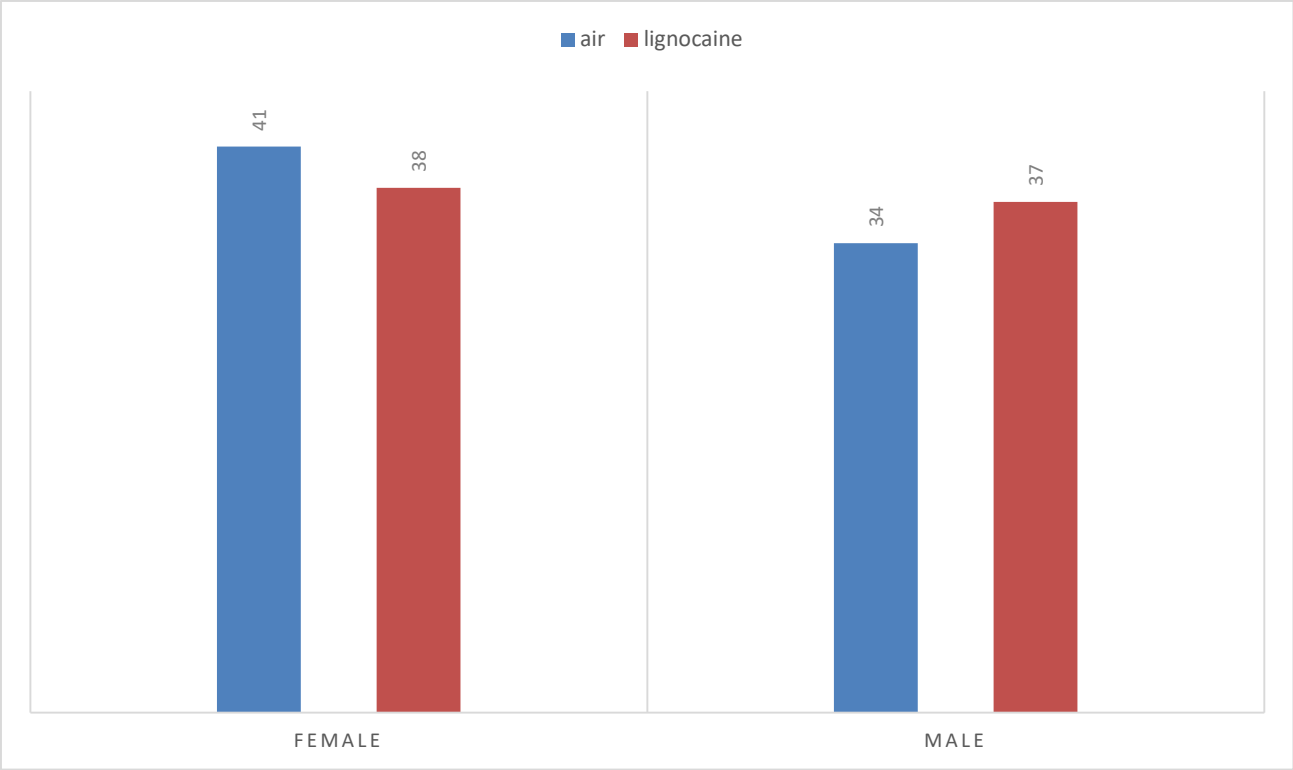


Figure 15:- Gender distribution of the sample

		cuff		Total
		air	Lignocaine	
SEX	Female	41 (54.7%)	38 (50.7%)	79
	Male	34 (45.3%)	37 (49.3%)	71
Total		75	75	150

Table 7:- Gender distribution among the samples

Distribution of sample according to planned surgical procedure :

The following figure shows distribution of patients in both the groups with respect to the surgical procedure .

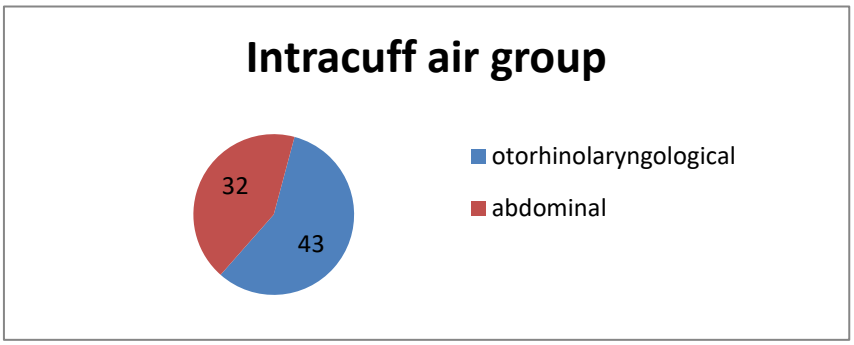


Fig 16:- Distribution of sample with respect to surgical procedure while using air as cuff inflation medium.

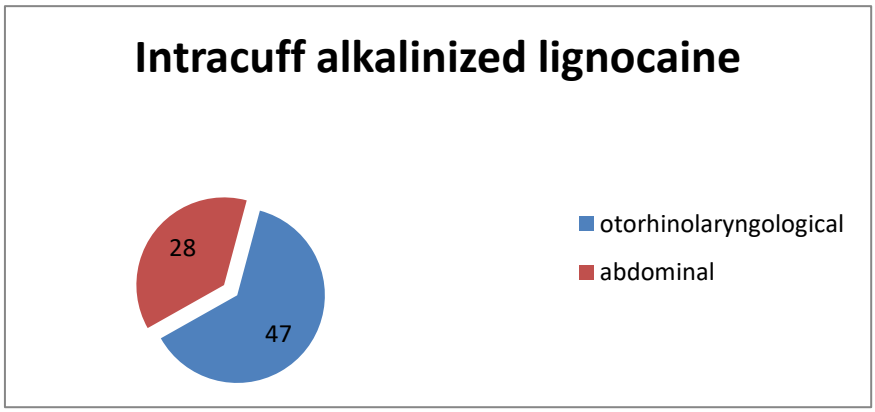


Fig 17:- Distribution of sample with respect to surgical procedure while using alkalinized lignocaine as cuff inflation medium.

ASA Physical status among the two groups

The following figures and tables show that there is no significant difference in terms of ASA physical status between both the groups.

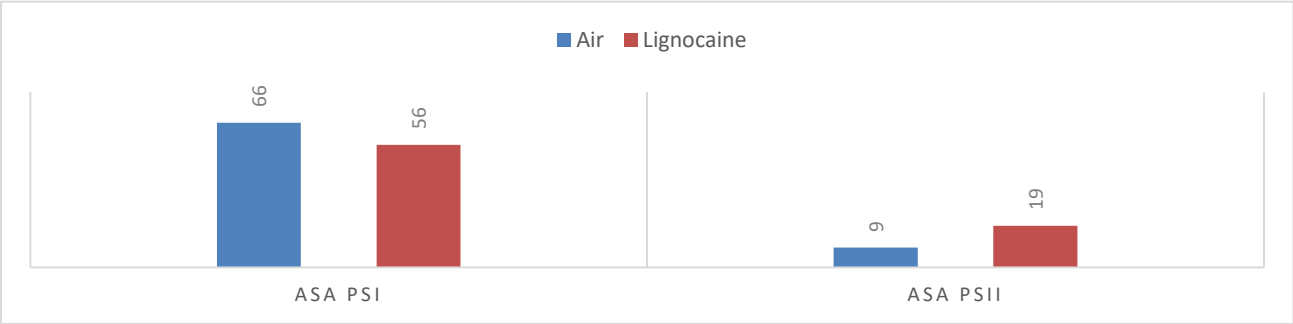


Figure 18: ASA Physical Status

		cuff		Total	
		air	lignocaine		
ASA Physical status	ASA PSI	66	56	122	
	ASA PSII	9	19	28	
Total		75	75	150	

Table 8:- Distribution of sample in terms of ASA physical status

Cuff pressure at the end of the surgery

The following figures show the cuff pressure at the start and at the end of the surgery. The cuff pressure at the end of surgery showed a one and a half to two fold increase compared to initial cuff pressure in the air group. However, the final cuff pressure in the lignocaine group was similar to their initial cuff pressures. The two groups differ significantly with a p value <0.05

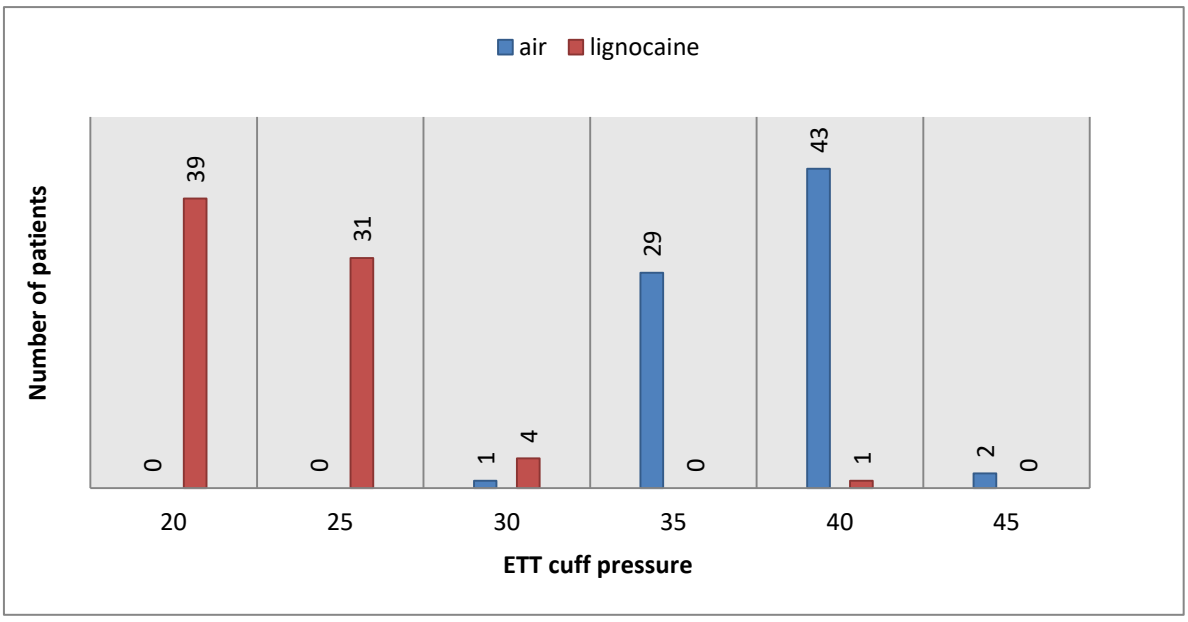


Fig 19:- Cuff pressure at the end of surgery

		Cuff		Total		
		air	Lignocaine		Chi square value	p value
cuff pressure (end)	20	0	39	39		
	25	0	31	31		
	30	1	4	5		
	35	29	0	29	142.891	<0.05
	40	43	1	44		
	45	2	0	2		
Total		75	75	150		

Table 9:- Cuff pressure at the end of the surgery

Post operative sore throat immediately after extubation :

The following tables and figures show the severity of sore throat immediately after extubation . Score 0 - no sore throat , Score 1 - minimal - patient answered in the

affirmative when asked about sore throat , Score 2 - moderate - patient complained of sore throat on his/her own

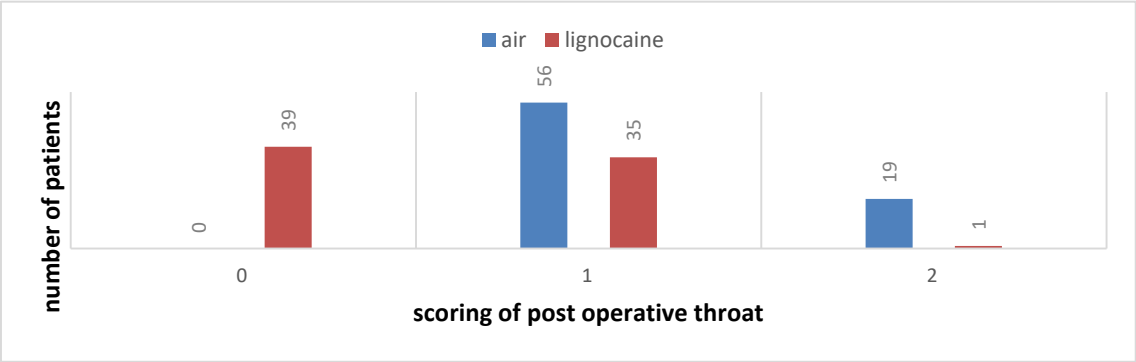


Figure 20:- Post operative Sore throat after extubation

		cuff		Total		
		air	lignocaine		Chi square value	p value
0 MIN	0	0	39	39		
	1	56	35	91	60.046	<0.05
	2	19	1	20		
Total		75	75	150		

Table 10:- Severity of sore throat after extubation

Immediately after extubation, the severity of post operative sore throat was more (score 1) in the air group compared to lignocaine group.

Sore throat at 1 hour after extubation :

The lignocaine group showed lesser severity of post operative sore throat compared to air group one hour after extubation.

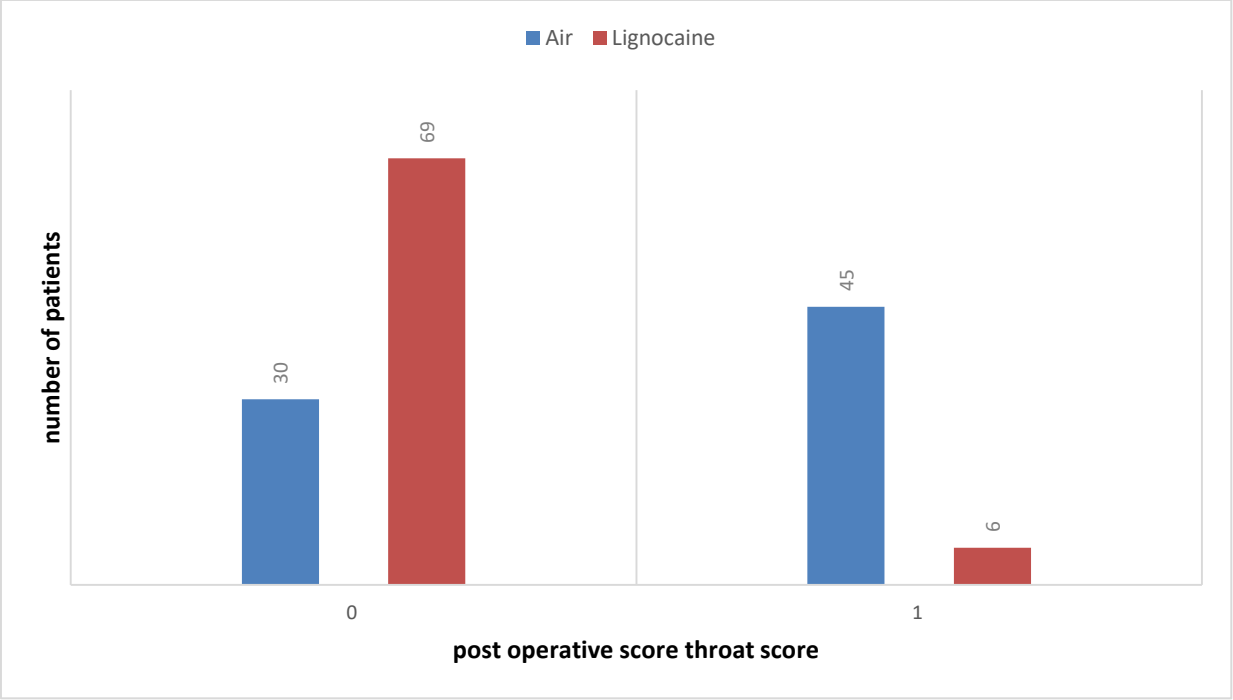


Figure 21:- Sore throat at 1 hour after extubation

		cuff		Total		
		Air	lignocaine		Chi square value	p value
1 HR	0	30	69	69		
	1	45	6	81	127.778	<0.05
Total		75	75	150		

Table 11 : Sore throat at 1 hour after extubation

Sore throat 6 hours after extubation :

The subjects belonging to both groups did not vary sugnificantly in terms of severity of post operative sore throat 6 hours after extubation.

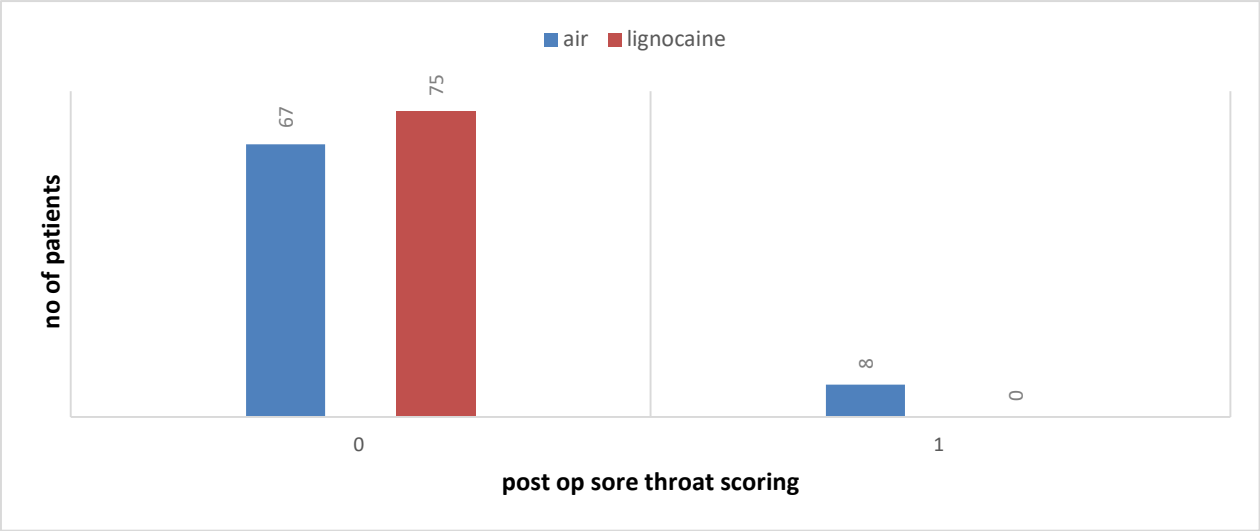


Figure 22:- Sore throat at 6 hours after extubation

		cuff		Total		
		air	lignocaine		Chi square value	p value
6HRS	0	67	75	142	8.451	>0.05
	1	8	0	8		
Total		75	75	150		

Table 12: Sore throat at 6 hours after extubation

Other features

The following table and figures show comparison of incidence of the other features like post extubation cough, nausea, vomiting and hoarseness of voice among the two groups. Nausea and vomiting was present in 91 patients (air=45; lignocaine=46). Statistical analysis shows that p>0.005 which is statistically insignificant.

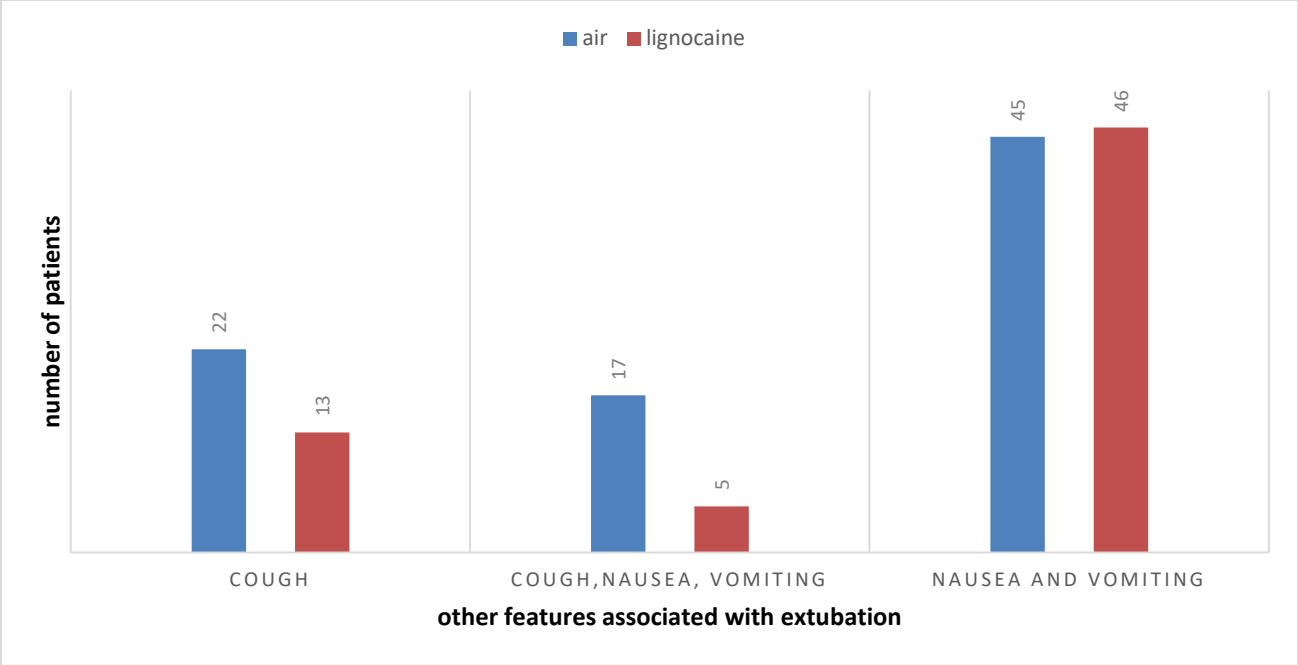


Fig 23:- Incidence of other features associated with extubation between air and lignocaine group

		cuff		Total	
		air	lignocaine		p value
OTHER FEATURES	cough	22	13	35	
	cough nausea vomiting	17	5	22	
	Nausea and vomiting	45	46	91	>0.05
	nil	0	2	2	
Total		75	75	150	

Table 13:- Other features associated with extubation

Differences between Male and female subjects among two groups with respect to sore throat just after extubation : (0 Min)

There was significant difference among males and females only in the air group with a p value <0.05. The following figures and tables show the differences

in the severity of post operative sore throat among males and females just after extubation.

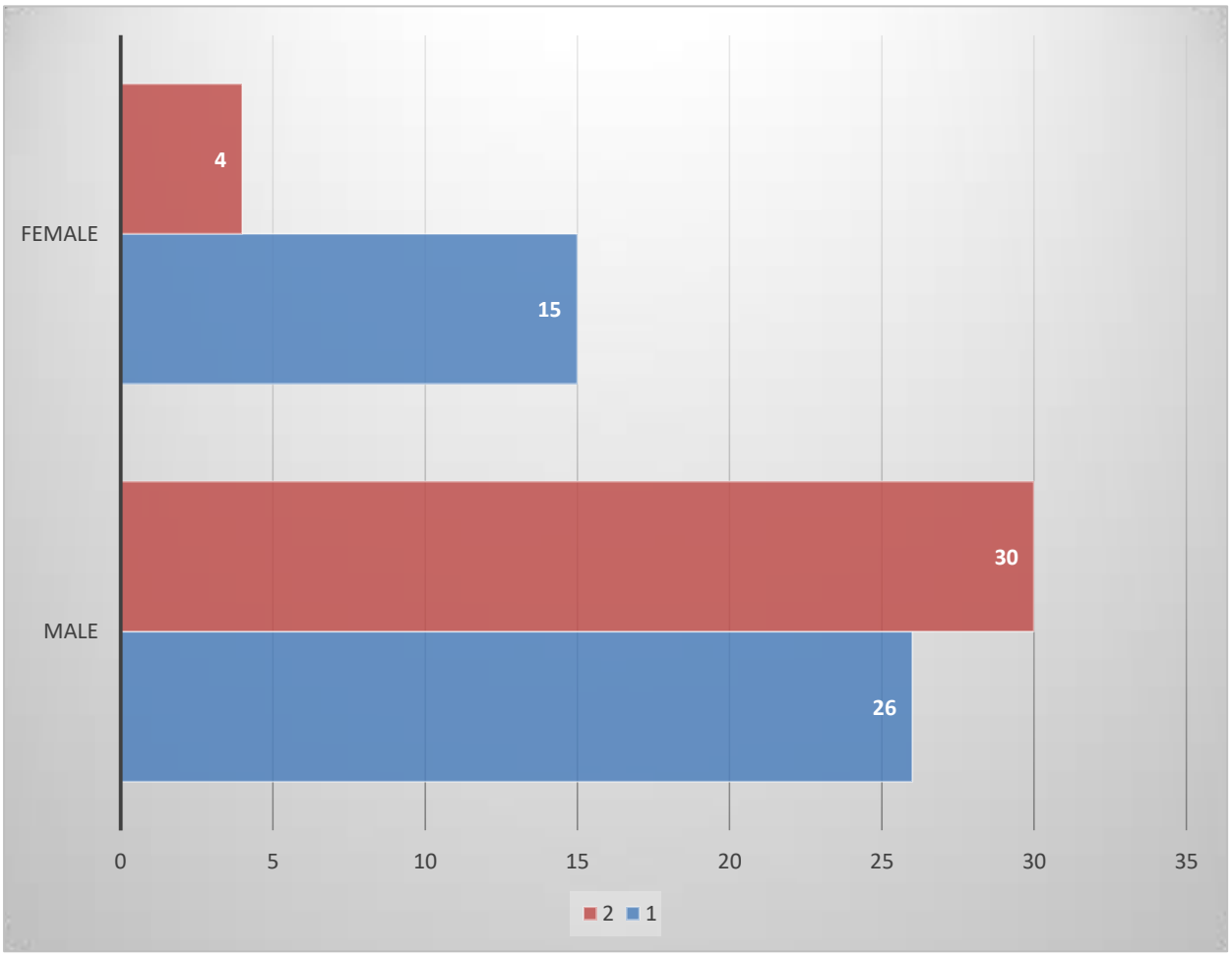


Figure 24:- Difference between male and female in intracuff Air group

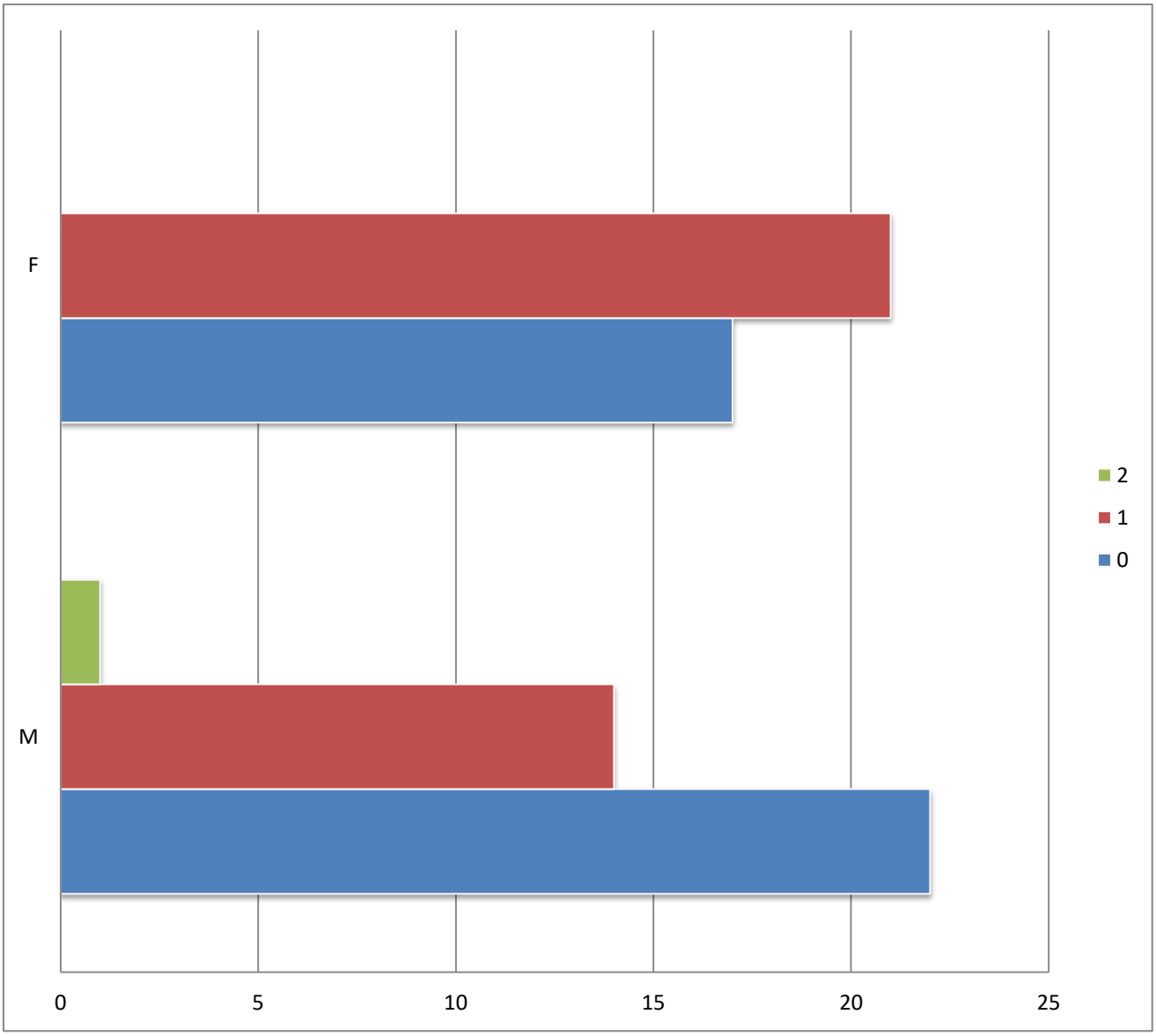


Figure 25:- Difference between male and female in intracuff alkalinized

Lignocaine group

Air		SEX		Total
		F	M	
0 MIN	1	26	30	56
	2	15	4	19
Total		41	34	75

Table 14: Difference in postoperative sorethroat severity between male and female in air group at 0 mins

Lignocaine		SEX		Total
		F	M	
0 MIN	0	17	22	39
	1	21	14	35
	2	0	1	1
Total		38	37	75

Table 15: Difference in postoperative severity between male and female in intracuff alkalinized Lignocaine group at 0 mins

CHAPTER 13

DISCUSSION

In our study comparing the effect alkalinized lignocaine and air as inflation media of the cuff it was found that using alkalinized lignocaine as the cuff inflation medium led to a significant reduction in the incidence of sore throat upon emergence from general anesthesia. Also, the incidence of other side effects associated with endotracheal intubation like postoperative cough , hoarseness of voice, PONV were also decreased in comparison to the group in which air was used as the cuff inflation media.

The distribution of the sample according to gender, age, ASA physical status and type of surgical procedure were comparable among both the groups.

In our clinical study, the cuff pressure was recorded at the start and end of the surgery in both groups. The initial cuff pressure was maintained at 20 cm H_2O in both the groups and the change in the cuff pressure towards the end of surgery were compared in both the groups. The Chi square test showed that the difference between the initial and final cuff pressure in the lignocaine group was statistically

significant with p value <0.05 compared to the air group. The final cuff pressure in air filled cuffs showed higher values ranging from 29- 45 cm H_2O implying diffusion of nitrous oxide throughout the duration of surgery.

However, the cuff pressure in the lignocaine group towards the end of surgery did not show a drastic increase from their initial pressures. An approximate increase of 25% in the final cuff pressure was noted.

The comparison of sore throat at various intervals after extubation among the the two groups were computed statistically using the Chi Square test. Immediately after extubation, the severity of post operative sore throat was more (score 1) in the air group compared to the lignocaine group , which proved to statistically significant (Chi square value - 60.046 , p value <0.05).

One hour after extubation, the severity of post operative sore throat was significantly less in the lignocaine group as compared to the air group. At 6 hours, 12 hours and 24 hours after extubation, subjects belonging to both the groups did not vary significantly in terms of severity of post operative sore throat.

Other features associated with emergence phenomenon like nausea, vomiting, cough and hoarseness of voice were also compared between the two groups. The incidence of cough was higher in the air group thus suggesting cough suppressant effect of lignocaine. This is in accordance with the study conducted by Fegan et al[49] which showed decreased incidence of cough 4 - 8 minutes after extubation.

However, the incidence of nausea, vomiting, and bucking was similar among both the groups. This is in contrast to the study conducted y Dr. Wasim Salman et al[50] which showed decreased incidence of nausea, vomiting, bucking and restlesness after extubation with intracuff lignocaine.

Our results showed that the male patients reported a higher incidence of post operative sore throat in the air as compared to their female counterparts which is most likely due to insertion of larger sized endotracheal tube. However, the severity of post operative throat in the lignocaine group immediately after extubation was more among female patients.

Earlier studies evaluating the efficacy of intracuff lidocaine regarding similar clinical outcomes (i.e., postoperative sore throat and coughing) used larger

quantities of non-alkalinized lidocaine (200-500 mg) in an attempt to increase its diffusion —possibly increasing the risk of toxicity in the event of cuff rupture. We now know that alkalinization reduces the required amount of intracuff lidocaine needed while maintaining its effectiveness.

In a study conducted by Souisii H et al[47], 160 mg of alkalinized lignocaine was used to inflate the cuff and it was found that significant amount of lignocaine diffused via the cuff in two hours. In a study by Shroff et al.[45], 40 mg of intracuff alkalinized lidocaine had no impact on cough at emergence compared with intracuffsaline.In contrast, using a larger amount of alkalinized lidocaine (200 mg), Huang et al[39]. observed a significant decrease in cough compared to saline. Even though it was tested in a population of smokers, similar effects of alkalinized lidocaine (138 ± 52 mg) vs saline were subsequently found by another group.

Considering its diffusion dynamics and clinical safety profile, we used 130 mg of alkalinized lidocaine as the cuff-filling medium in this clinical study. Nitrous oxide usage during anesthetic management of patients is associated with cuff over-inflation, which in turn is associated with damage to the pharyngeal mucosa.

The use of intracuff liquids is known to prevent overinflation by limiting the diffusion of N2O inside the cuff. In these specific anesthetic conditions, prevention of over-inflation by any cuff-filling liquids (e.g., saline or alkalinized lidocaine vs air in the cuff) may therefore be a more likely mechanism to explain the reduced incidence of sore throat, in contrast to the potential local anesthetic effect of lidocaine. Hence, monitoring of endotracheal tube cuff pressure during general anaesthesia is of paramount importance .

In fact, lidocaine diffusion from an ETT cuff inflated farther away from the vocal cord may produce a cough suppressant effect .In our clinical trial, only patients whose surgery was estimated to last 120 min or more were enrolled. Our rationale was that this period would provide sufficient diffusion time to obtain an effective local concentration of anesthetic to block the rapidly adapting stretch receptors, located within the tracheal mucosa, that are responsible for the cough reflex. Animal studies suggest that a lidocaine concentration of 155 mg/mL is necessary to block these receptors. Other potentially confounding factors (including narcotics, non-steroidal anti-inflammatory drugs, time under anesthesia) were similar in the two treatment groups. We found no evidence of aspiration either of

the groups. Our results could be generalized to other surgery of the same average duration for adult patients of both sexes.

CHAPTER 14

CONCLUSION

In our study we conclude that inflating the cuff of the endotracheal tube with alkalinized lidocaine avoids the cuff overinflation due to rapid trans-cuff N_2O diffusion during general anesthesia with nitrous-oxygen mixture. Hence, inflating the endotracheal tube cuff with alkalinized lignocaine is an alternative method to prevent post operative sore throat and other potential complications of extubation including post operative cough and hoarseness of voice which results from cuff overinflation in patients undergoing general anaesthesia.

CHAPTER 15

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ANNEXURE

QUESTIONAIRE

PATIENT DETAILS:

Name: Age: Sex:

IP No. :

CLINICAL DIAGNOSIS:

SURGERY PLANNED :

PRE OPERATIVE ASSESSMENT :

AIRWAY :	DENTITION :	NECK :	SPINE :
Pulse :	BP :	RR :	Temp :
CVS :	RS :		
P/A :	CNS:		

INVESTIGATIONS :

Hb% TC Platelets:

RBS: Urea: Creatinine: Na^+ :K^+:

CXR :

ECG :

HISTORY OF RECENT RESPIRATORY TRACT INFECTION :

HISTORY OF PREVIOUS SURGERY DONE UNDER GENERAL ANAESTHESIA:

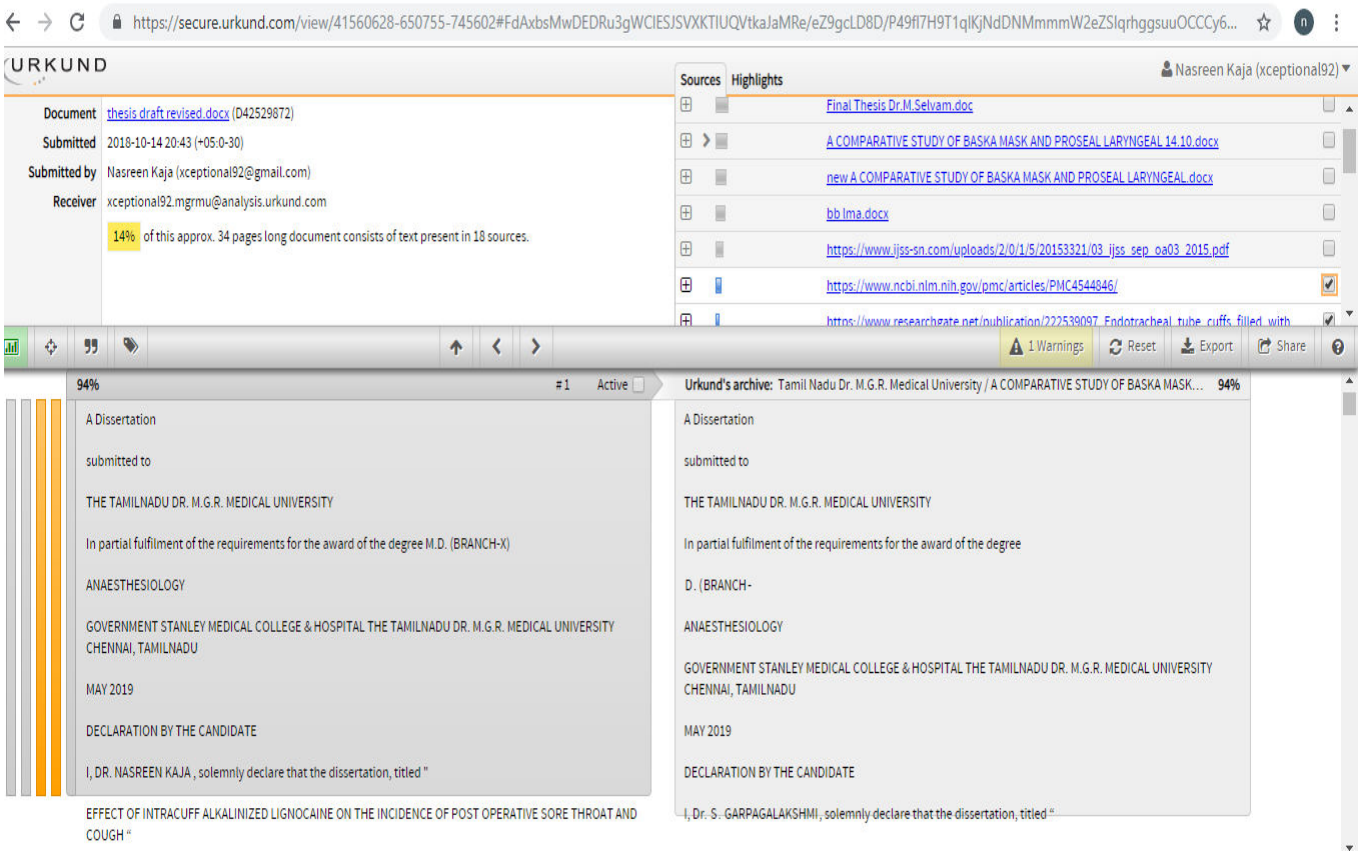
HISTORY OF DRUG INTAKE :

PATIENT ASSESSED UNDER ASA PS –

GRADING OF POST OPERATIVE SORE THROAT

FEATURE			SCORE		
			DURATION AFTER EXTUBATION		
	0 min	1 hour	6hrs	12hrs	24 hours
No sore throat at any time since the surgery (0)					
Minimal – patient answered in the affirmative when asked about sore throat (1)					
Moderate – patient complained of sore throat on his/her own (2)					
Severe- patient is in obvious distress (3)					

FEATURE	PRESENT / ABSENT
Post operative cough	
Nausea and vomiting	
Hoarseness of voice	
Evidence of Aspiration	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In partial fulfilment of the requirements for the award of the degree M.D. (BRANCH-X)
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GOVERNMENT STANLEY MEDICAL COLLEGE & HOSPITAL THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY CHENNAI, TAMILNADU
MAY 2019
DECLARATION BY THE CANDIDATE
I, DR. NASREEN KAJA , solemnly declare that the dissertation, titled "
EFFECT OF INTRACUFF ALKALINIZED LIGNOCAINE ON THE INCIDENCE OF POST OPERATIVE SORE THROAT AND COUGH "
Urkund's archive: Tamil Nadu Dr. M.G.R. Medical University / A COMPARATIVE STUDY OF BASKA MASK... 94%
A Dissertation
submitted to
THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY
In partial fulfilment of the requirements for the award of the degree
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MAY 2019
DECLARATION BY THE CANDIDATE
I, Dr. S. GARPAGALAKSHMI , solemnly declare that the dissertation, titled "

INFORMATION SHEET

TITLE :"EFFECT OF INTRACUFF ALKALINIZED LIGNOCAINE ON THE INCIDENCE OF POST OPERATIVE SORE THROAT AND COUGH"

Name of Investigator : Dr. NASREEN KAJA , Name of Participant :

Purpose of Research : To prevent post operative sore throat and cough after general anaesthesia.

Study Design : Prospective Study.

Study Procedures : Patient will be subjected to general anesthesia with the cuff of the endotracheal tube being inflated with alkalinized lignocaine.

Possible Risks : No risks to the patient

Possible benefits

To patient : A better understanding of their problem so as to devise a plan of management which suits their needs.

To doctor & to other people : Based upon the study, post operative sore throat and cough after general anaesthesia can be reduced.

Confidentiality of the information obtained from you : The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Can you decide to stop participating in the study : Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time

How will your decision to not participate in the study affect you : Your decision will not result in any loss of benefits to which you are otherwise entitled.

Signature of Investigator Signature of Participant

Date :

Place :

PATIENT CONSENT FORM

Study Detail : "EFFECT OF INTRACUFF ALKALINIZED LIGNOCAINE ON THE INCIDENCE OF POST OPERATIVE SORE THROAT AND COUGH"

Study Centre : Govt. Stanley medical college , Chennai.

Patient's Name :

Patient's Age :

In Patient Number :

Patient may check (☑) these boxes

I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction. ❑

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected. ❑

I understand that sponsor of the clinical study, others working on the sponsor's behalf, the Ethics committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study. ❑

I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well being or any unexpected or unusual symptoms. ❑

I hereby consent to participate in this study ❑

I hereby give permission to undergo complete clinical examination and diagnostic tests including hematological, biochemical, radiological tests and to undergo treatment ❑

Signature/thumb impression

Patient's Name and Address:

Signature of Investigator

Study Investigator's Name:

Dr.NASREEN KAJA

QUESTIONAIRE

PATIENT DETAILS:

Name: Age: Sex:

IP No. :

CLINICAL DIAGNOSIS:

SURGERY PLANNED :

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P/A : CNS:

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Hb% TC Platelets:

RBS: Urea: Creatinine: Na^+ :K^+:

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No sore throat at any time since the surgery (0)					
Minimal – patient answered in the affirmative when asked about sore throat (1)					
Moderate – patient complained of sore throat on his/her own (2)					
Severe- patient is in obvious distress (3)					

FEATURE	PRESENT / ABSENT
Post operative cough	
Nausea and vomiting	
Hoarseness of voice	
Evidence of Aspiration	

INFORMED CONSENT

A STUDY ON THE EFFECT OF INTRACUFF ALKALINIZED LIGNOCAINE ON THE INCIDENCE OF POST OPERATIVE SORE THROAT AND COUGH
AT GOVERNMENT STANLEY HOSPITAL, CHENNAI.

நான்இந்தஆராய்ச்சியில்விவரங்களைமுற்றிலும்புரிந்துகொண்டேன்.
ஆய்வில்பங்குஎடுத்துபோது,
சாத்தியமானஅபாயங்கள்மற்றும்பயன்களைபற்றிநான்அறிந்துள்ளேன்.
நான்எந்தவொருவேளையிலும்ஆய்வில்இருந்துதிரும்பமுடியும், அதன்பின்னர்,
நான்வழக்கம்போல்மருத்துவசிகிச்சைபெறமுடியும்என்றுபுரிந்துகொள்கிறேன்
நான்ஆய்வில்பங்குஎடுத்துபணம்எதையும்பெறமுடியாதுஎன்றுஅறிந்துள்ளேன்.
இந்தஆய்வின்முடிவுகள்எந்தமெடிக்கல்ஜர்னலில்வெளியிடப்படஇருந்தால்நான்எதிர்க்கவில்லை, என்தனிப்பட்டஅடையாளத்தைவெளிப்படுத்தப்பட்டுஇருக்ககூடாது.
நான்இந்தஆய்வில்பங்கெடுப்பதன்மூலம்நான்என்னசெய்யபோகிறேன்என்றுதெரியும்
நான்இந்தஆய்வில்என்முழுஒத்துழைப்பையும்கொடுப்பேன்என்றுஉறுதியளிக்கிறேன்.

தன்னார்வளர் சாட்சி
பெயர்மற்றும்முகவரி பெயர்மற்றும்முகவரி
கையொப்பம் / விரல்ரேகை: கையொப்பம் / விரல்ரேகை:

ஆராய்ச்சியாளராக கையொப்பம்மற்றும்தேதி

ANTI PLAGIARISM CERTIFICATE

This is to certify that this dissertation work titled A study on The **Effect of Intracuff Alkalinized Lignocaine On The Incidence of Post Operative Sore Throat and Cough** of the candidate **Dr. NASREEN KAJA** with registration Number**201620054**for the award of **M.D ANAESTHESIOLOGY** in the branch of **X.** I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows 14%.percentage of plagiarism in the dissertation.

Guide & Supervisor sign with Seal.

GOVERNMENT STANLEY MEDICAL COLLEGE & HOSPITAL,CHENNAI -01
INSTITUTIONAL ETHICS COMMITTEE

TITLE OF THE WORK : EFFECT OF INTRACUFF ALKALINIZED LIGNOCAINE ON THE INCIDENCE OF POST OPERATIVE SORE THROAT AND COUGH.

PRINCIPAL INVESTIGATOR : DR. NASREEN KAJA,
DESIGNATION : PG IN MD ANAESTHESIOLOGY,
DEPARTMENT : DEPARTMENT OF ANAESTHESIOLOGY, GOVT. STANLEY MEDICAL COLLEGE.

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 13.04.2018 at the Council Hall, Stanley Medical College, Chennai-1 at 10am.

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

MEMBER SECRETARY,
IEC, SMC, CHENNAI

NAME	AGE/SEX	IP. NO.	DIAGNOSIS	PROCEDURE DONE	cuf pressure (end)	0 MIN	1 HR	6HRS	12HRS	24HRS	OTHER FEATURES	cuff	ASA Physical status
priyanka	19/f	300343	CSOM	tympanoplasty	25	1	0	0	0	0	nausea and vomiting	lignocaine	ASA PSI
prakasam	56/m	188762	CSOM	cortical mastoidectomy	20	0	0	0	0	0	cough	lignocaine	ASA PSI
desamuthu	50/f	1936895	DNS	FESS	25	1	0	0	0	0	nausea and vomiting	lignocaine	ASA PSI
baskar	42/m	1314078	sinonasal polyposis	FESS	20	2	1	0	0	0	cough , nausea vomiting	lignocaine	ASA PSI
dilli babu	19/m	1914023	DNS	FESS	20	0	0	0	0	0	cough	lignocaine	ASA PSI
jegankumar	38/m	1924304	DNS	FESS	25	0	0	0	0	0	nausea and vomiting	lignocaine	ASA PSI
vijaykumar	19/m	1884555	CSOM	tympanoplasty	20	1	1	0	0	0	nausea and vomiting	lignocaine	ASA PSI
livingston	18/m	1927016	CSOM	cortical mastoidectomy	25	1	0	0	0	0	Nausea and vomiting and cough	lignocaine	ASA PSI
venkatesh	24/m	1913344	DNS	FESS	20	0	0	0	0	0	Nausea and vomiting and cough	lignocaine	ASA PSI
ajith	22/m	1919529	DNS	FESS	20	1	0	0	0	0	nausea and vomiting	lignocaine	ASA PSI
umapathy	40/m	1923031	chronic sinusitis	FESS	20	1	1	0	0	0	cough nausea vomiting	lignocaine	ASA PSII
komaladevi	24/f	1916879	DNS	FESS	20	0	0	0	0	0	cough nausea vomiting	lignocaine	ASA PSII
nagavalli	29/f	309055	DNS	FESS	25	1	0	0	0	0	nausea and vomiting	lignocaine	ASA PSII
saradha	38/f	1934255	chronic sinusitis	FESS	20	1	0	0	0	0	cough	lignocaine	ASA PSII
pandiyan rajan	38/m	1948565	DNS	FESS	25	1	1	0	0	0	nausea and vomiting	lignocaine	ASA PSII
shalini	20/f	1838163	DNS	FESS	20	0	0	0	0	0	nausea and vomiting	lignocaine	ASA PSII
divyashree	23/f	1875737	DNS	FESS	20	1	0	0	0	0	nausea and vomiting	lignocaine	ASA PSII
sagila	32f	1910408	CSOM	cortical mastoidectomy	20	0	0	0	0	0	nausea and vomiting	lignocaine	ASA PSII
azhagavel	38m	861852	DNS	FESS	30	1	0	0	0	0	nausea and vomiting	lignocaine	ASA PSII
shobiya	19f	857137	DNS	FESS	20	0	1	0	0	0	nil	lignocaine	ASA PSII
shantipriya	24f	1964535	DNS	FESS	25	1	0	0	0	0	nausea and vomiting	lignocaine	ASA PSII
ibrahim	27m	1847959	DNS	FESS	20	0	0	0	0	0	nausea and vomiting	lignocaine	ASA PSII
kanniyammal	54f	1967708	sinonasal polyposis	fess	25	1	0	0	0	0	nausea and vomiting	lignocaine	ASA PSII
muhukumar	39m	1845023	DNS	FESS	20	1	0	0	0	0	nausea and vomiting	lignocaine	ASA PSII
rajendran	42m	1847959	DNS	FESS	25	0	0	0	0	0	nausea and vomiting	lignocaine	ASA PSII
radhika	29f	1954635	DNS	FESS	20	1	0	0	0	0	nil	lignocaine	ASA PSII
selvi	32f	1963955	CSOM	cortical mastoidectomy	20	1	0	0	0	0	nausea and vomiting	lignocaine	ASA PSII
kali	56m	1807404	inguinal hernia	lap hernia	20	1	0	0	0	0	nausea and vomiting	lignocaine	ASA PSII
sumathy	31f	1808839	cholelithiasis	lap cholecystectomy	30	0	0	0	0	0	nausea and vomiting	lignocaine	ASA PS I
selvi	40f	1802278	cholelithiasis	lap cholecystectomy	20	0	0	0	0	0	nausea and vomiting	lignocaine	ASA PS I
banumathy	63f	1890108	cholelithiasis	lap cholecystectomy	25	0	0	0	0	0	nausea and vomiting	lignocaine	ASA PS I
sridhar	53m	1772710	pleomorphic adenoma	superficial parotidectomy	20	1	0	0	0	0	nausea and vomiting	lignocaine	ASA PS I
viswanathan	68m	1804178	cholelithiasis	lap cholecystectomy	20	0	0	0	0	0	cough	lignocaine	ASA PS I
janaki	28f	1805467	gb polyp	lap cholecystectomy	20	1	0	0	0	0	cough	lignocaine	ASA PS I
ponniyan selvi	62f	1801639	ca breast	mrm	20	1	1	0	0	0	cough	lignocaine	ASA PS I
indumathy	37f	1807211	ca breast	lumpectomy	20	0	0	0	0	0	cough	lignocaine	ASA PS I
kulandaram	52f	1806222	ca breast	mrm	20	1	0	0	0	0	nausea and vomiting	lignocaine	ASA PS I
senthil	40m	1821300	inguinal hernia	lap hernia	25	0	0	0	0	0	nausea and vomiting	lignocaine	ASA PS I I
devaki	60f	1818841	ovarian cyst	d lap	20	1	0	0	0	0	nausea and vomiting	lignocaine	ASA PS I
murugaperumal	44m	1810594	incisional hernia	mesh repair	25	0	0	0	0	0	nausea and vomiting	lignocaine	ASA PS I
mohanraj	60m	1819146	cholelithiasis	lap cholecystectomy	20	0	0	0	0	0	nausea and vomiting	lignocaine	ASA PS I
salima	35f	1819382	cholelithiasis	lap cholecystectomy	25	1	0	0	0	0	nausea and vomiting	lignocaine	ASA PS I
selvi	38f	1809037	adnexal mass	d lap	20	0	0	0	0	0	nausea and vomiting	lignocaine	ASA PS I
indumathy	45f	1811437	ca breast	mrm	25	1	0	0	0	0	nausea and vomiting	lignocaine	ASA PS I
indumathy	30f	1812500	cholelithiasis	lap cholecystectomy	25	0	0	0	0	0	nausea and vomiting	lignocaine	ASA PS I
amsa	59f	1812528	cholelithiasis	lap cholecystectomy	25	0	0	0	0	0	nausea and vomiting	lignocaine	ASA PS I
rubavathy	32f	1810000	cholelithiasis	lap cholecystectomy	25	0	0	0	0	0	nausea and vomiting	lignocaine	ASA PS I
shankar	43m	1812773	cholelithiasis	lap cholecystectomy	25	0	0	0	0	0	cough	lignocaine	ASA PS I
gandimathy	61f	1807770	cholelithiasis	lap cholecystectomy	25	1	0	0	0	0	cough	lignocaine	ASA PS I

ramanujam	50m	1823977	inguinal hernia	lap hernia	30	0	0	0	0	0	cough	lignocaine	ASA PS I
abdul rafi	71m	1820890	inguinal hernia	lap hernia	20	0	0	0	0	0	cough	lignocaine	ASA PS I
sangara reddy	61m	1822654	cholelithiasis	lap cholecystectomy	20	0	0	0	0	0	cough	lignocaine	ASA PS I
sumathy	43f	1827586	incisional hernia	mesh repair	25	1	0	0	0	0	cough	lignocaine	ASA PS I
maheshwari	60f	1802692	cholelithiasis	lap cholecystectomy	20	0	0	0	0	0	cough	lignocaine	ASA PS I
noorjahan	48f	1825692	cholelithiasis	lap cholecystectomy	20	0	0	0	0	0	cough	lignocaine	ASA PS I
naveenkumar	27m	1826136	gynaecomastia	websters procedure	25	1	0	0	0	0	cough	lignocaine	ASA PS I
josna	42f	1826139	parotid sialadenitis	superficial parotidectomy	25	0	0	0	0	0	cough	lignocaine	ASA PS I
rameela	28f	1804311	cholelithiasis	lap cholecystectomy	20	0	0	0	0	0	nausea and vomiting	lignocaine	ASA PS I
sarain kumar	25m	1832108	inguinal hernia	lap hernia	25	0	0	0	0	0	nausea and vomiting	lignocaine	ASA PS I
murugan	54m	1830563	inguinal hernia	lap hernia	20	1	0	0	0	0	nausea and vomiting	lignocaine	ASA PS I
kamalnathan	45m	1882097	cholelithiasis	lap cholecystectomy	25	0	0	0	0	0	nausea and vomiting	lignocaine	ASA PS I
arifa	16f	1833776	abdominal pain for evaluation	d lap	20	1	0	0	0	0	nausea and vomiting	lignocaine	ASA PS I
elangovan	30m	1832070	cholelithiasis	lap cholecystectomy	25	0	0	0	0	0	nausea and vomiting	lignocaine	ASA PS I
vajravel	57m	1830591	incisional hernia	mesh repair	20	0	0	0	0	0	nausea and vomiting	lignocaine	ASA PS I
hajira	48f	1840591	CSOM	cortical mastoidectomy	30	1	0	0	0	0	nausea and vomiting	lignocaine	ASA PS I
dhanasekhar	62m	1855963	inguinal hernia	lap hernia	25	0	0	0	0	0	nausea and vomiting	lignocaine	ASA PS I
ramamoorthy	49m	1857938	inguinal hernia	lap hernia	25	0	0	0	0	0	nausea and vomiting	lignocaine	ASA PS I
ezhilarasan	30m	1860736	gynaecomastia	websters procedure	25	0	0	0	0	0	cough	lignocaine	ASA PS I
selvaraj	42m	1855305	cholelithiasis	lap cholecystectomy	20	1	0	0	0	0	cough	lignocaine	ASA PS I
govindaraj	38m	1856417	incisional hernia	mesh repair	25	0	0	0	0	0	cough	lignocaine	ASA PS I
rajeshwari	26f	1805427	DNS	FESS	20	1	0	0	0	0	cough	lignocaine	ASA PS I
suresh kumar	34m	1693393	DNS	FESS	25	1	0	0	0	0	cough	lignocaine	ASA PS I
sasikala	42f	1808829	chronic sinusitis	FESS	20	1	0	0	0	0	nausea and vomiting	lignocaine	ASA PS I
vijaya	49f	1808293	sinonasal polyposis	FESS	25	0	0	0	0	0	nausea and vomiting	lignocaine	ASA PS I
shathikali	37m	1807837	CSOM	cortical mastoidectomy	40	1	0	0	0	0	nausea and vomiting	lignocaine	ASA PS I
Anbarasan	46m	1772748	recurrent submandibular sialad	submanidibular gland excision	35	2	1	0	0	0	cough	air	ASA PS I
Mary	60f	1174261	csom	cortical mastoidectomy	35	2	1	0	0	0	Nausea and vomiting	air	ASA PS I
Devi	36f	1731845	csom	cortcal mastoidectomy	40	1	1	0	0	0	Nausea and vomiting	air	ASA PS I
Keerthika	20f	1735319	DNS	Septal correction	40	1	1	0	0	0	Nausea and vomiting	air	ASA PS I
Decnan	42m	1686101	B/L oto scelerosis	L stapedectomy	35	1	1	0	0	0	Nausea and vomiting	air	ASA PS I
Lalitha	30f	1706813	DNS	FESS	40	2	1	0	0	0	Nausea and vomiting	air	ASA PS I
Kumaresan	38m	1724676	sinonasal polyposis	FESS	35	1	1	0	0	0	Nausea and vomiting	air	ASA PS I
Kailasam	29m	1810765	facial nerve palsy	facial decompression	40	1	1	0	0	0	Nausea and vomiting	air	ASA PS I
Jeyanthi	48f	181043	csom	cortical mastoidectomy	45	2	1	0	0	0	Nausea and vomiting and cough	air	ASA PS I
Vasanthi	24f	839536	csom	tympanoplasty	40	2	1	0	0	0	Nausea and vomiting	air	ASA PS I
Pushpa	18f	1706108	chronic sinusitis	FESS	40	2	1	0	0	0	Nausea and vomiting	air	ASA PS I
Saranya	25f	1725901	DNS	FESS	35	1	1	0	0	0	Nausea and vomiting and cough	air	ASA PS I
Regina	16f	1610391	DNS	FESS	40	1	1	0	0	0	Nausea and vomiting	air	ASA PS I
surya	19m	51693	DNS	FESS	40	1	1	1	0	0	Nausea and vomiting	air	ASA PS I
sophiya	22f	842692	csom	tympanoplasty	40	1	1	0	0	0	Nausea and vomiting	air	ASA PS I
babu	25m	1516837	csom	cortical mastoidectomy	40	1	1	0	0	0	Nausea and vomiting	air	ASA PS I I
arun	19m	1809444	chronic sinusitis	FESS	40	1	1	1	0	0	Nausea and vomiting and cough	air	ASA PS I I
abdul mubarak	28m	1709882	DNS	FESS	40	2	1	0	0	0	Nausea and vomiting	air	ASA PS I
joseph	32m	162813	DNS	FESS	35	1	1	0	0	0	Nausea and vomiting	air	ASA PS I
mohan	18m	180877	csom	cortical mastoidectomy	40	1	1	0	0	0	Nausea and vomiting	air	ASA PS I
selvi	36f	1640342	otosclerosis	tympanoplasty	35	2	1	0	0	0	Nausea and vomiting	air	ASA PS I
Anbarasan	29m	164321	csom	cortical mastoidectomy	40	1	1	0	0	0	Nausea and vomiting	air	ASA PS I
abdl rahim	19m	841229	DNS	FESS	45	1	1	0	0	0	Nausea and vomiting and cough	air	ASA PS I
hemalatha	44f	1816091	csom	cortical mastoidectomy	30	1	1	0	0	0	Nausea and vomiting and cough	air	ASA PS I
arif basha	17m	1703984	DNS	FESS	40	2	1	0	0	0	Nausea and vomiting	air	ASA PS I I

shafi	37m	1716475	otosclerosis	tympanoplasty	35	1	1	0	0	0	Nausea and vomiting and cough	air	ASA PS I
mariyammal	38f	1676817	DNS	FESS	40	1	1	0	0	0	Nausea and vomiting	air	ASA PS I
krishnaveni	28f	1645069	DNS	FESS	35	1	1	0	0	0	Nausea and vomiting and cough	air	ASA PS I
lathapriya	22f	1710010	csom	cortical mastoidectomy	40	2	1	0	0	0	Nausea and vomiting and cough	air	ASA PS I
lavanya	35f	1808046	csom	tympanoplasty	35	1	1	0	0	0	cough	air	ASA PS I
vasanthi	34f	1773285	DNS	FESS	40	1	1	0	0	0	Nausea and vomiting	air	ASA PS I
yasmin banu	28f	1640148	DNS	FESS	35	1	1	0	0	0	Nausea and vomiting and cough	air	ASA PS I
mohana	19f	839769	DNS	FESS	40	1	1	0	0	0	cough	air	ASA PS I
mary	43f	8281125	csom	cortical mastoidectomy	35	2	1	0	0	0	cough	air	ASA PS I
revathy	32f	1633102	ac polyp	FESS	40	1	1	0	0	0	cough	air	ASA PS I
anbarasu	46m	1772748	submandibular calculi	submanidibular gland excision	40	1	1	0	0	0	cough	air	ASA PS I I
balu	46m	810397	csom	cortical mastoidectomy	40	1	1	0	0	0	Nausea and vomiting and cough	air	ASA PS I
alamelu	30f	1634658	csom	cortical mastoidectomy	40	2	1	0	0	0	cough	air	ASA PS I
nandhakumar	24m	1629798	DNS	FESS	35	1	1	0	0	0	cough	air	ASA PS I
eswari	50f	1648212	csom	cortical mastoidectomy	40	1	1	0	0	0	Nausea and vomiting and cough	air	ASA PS I
sivakumar	22m	166488	csom	cortical mastoidectomy	35	1	1	1	0	0	Nausea and vomiting	air	ASA PS I I
mahalakshmi	24f	1809266	DNS	feSS	40	2	1	0	0	0	Nausea and vomiting and cough	air	ASA PS I
pechimuthu	36m	1809389	DNS	FESS	35	1	1	0	0	0	cough	air	ASA PS I
kiruba	40m	1726450	DNS	FESS	40	1	1	0	0	0	Nausea and vomiting and cough	air	ASA PS I
logesh	26m	1809446	DNS	feSS	40	1	1	0	0	0	Nausea and vomiting and cough	air	ASA PS I
nirmala	44f	1709219	DNS	FESS	40	2	1	0	0	0	Nausea and vomiting and cough	air	ASA PS I
sudha	27m	811803	csom	tympanoplasty	40	1	1	1	0	0	Nausea and vomiting	air	ASA PS I
komathy	32f	1685446	csom	cortical mastoidectomy	40	1	1	0	0	0	Nausea and vomiting	air	ASA PS I
ramu	48m	1648212	csom	cortical mastoidectomy	40	1	1	0	0	0	Nausea and vomiting	air	ASA PS I
fathima	44f	1805857	csom	cortical mastoidectomy	40	1	1	0	0	0	cough	air	ASA PS I
subashini	29f	1615210	dns	FESS	35	2	1	0	0	0	Nausea and vomiting	air	ASA PS I
kairunnisa	28f	1843219	DNS	FESS	35	1	1	1	0	0	Nausea and vomiting	air	ASA PS I
abdul sardar	50 m	1807223	csom	cortical mastoidectomy	35	1	1	0	0	0	Nausea and vomiting	air	ASA PS I I
hidayathulla	48m	176653	csom	tympanoplasty	35	1	1	0	0	0	Nausea and vomiting	air	ASA PS I
manjula	50f	1803719	polyp	FESS	40	1	1	0	0	0	cough	air	ASA PS I
vasanthy	33f	1635868	chronic sinusitis	FESS	35	2	1	0	0	0	Nausea and vomiting	air	ASA PS I
mahariba	36f	1362721	csom	cortical mastoidectomy	40	1	1	0	0	0	Nausea and vomiting	air	ASA PS I
velu	45m	1803178	sinonasal polyposis	FESS	35	1	1	1	0	0	Nausea and vomiting	air	ASA PS I
logeshwari	33f	1863180	csom	cortical mastoidectomy	40	1	1	0	0	0	Nausea and vomiting	air	ASA PS II
govindammal	38f	1803177	csom	cortical mastoidectomy	40	1	1	0	0	0	Nausea and vomiting	air	ASA PS I
ramu	34m	180160	facial nerve palsy	facial nerve decompression	35	1	1	0	0	0	Nausea and vomiting	air	ASA PS I
desarani	20f	1651565	csom	cortical mastoidectomy	40	2	1	0	0	0	Nausea and vomiting	air	ASA PS I
kadar basha	33m	1648463	dns	FESS	35	1	1	0	0	0	cough	air	ASA PS I
sethuraman	18m	1640658	DNS	feSS	40	1	1	1	0	0	Nausea and vomiting	air	ASA PS I
mary	43f	800484	otosclerosis	stapedotomy	35	1	1	0	0	0	nausea and vomiting	air	ASA PS I
selvi	37f	1678708	csom	cortical mastoidectomy	40	1	1	1	0	0	nausea and vomiting	air	ASA PS II
saravanan	23m	1662982	csom	cortical mastoidectomy	35	1	1	0	0	0	Nausea and vomiting	air	ASA PS I
balkis	40f	830416	csom	myringoplasty	40	1	1	0	0	0	Nausea and vomiting	air	ASA PS I
indhumathy	46f	1638526	ac polyp	FESS	35	2	1	0	0	0	Nausea and vomiting and cough	air	ASA PS I
kalaiarasi	25f	1849306	renal pelvic calculus	pcnl	40	1	1	0	0	0	cough	air	ASA PS I
ravi	40m	1851672	puj calculus	pcnl	35	1	1	0	0	0	Nausea and vomiting	air	ASA PS I
gunasekaran	50m	1853200	renal pelvic calculus	pcnl	40	2	1	0	0	0	Nausea and vomiting	air	ASA PS I
manikam	28m	1854517	staghorn calculus	pcnl	35	1	1	0	0	0	Nausea and vomiting and cough	air	ASA PS I
ajay	18m	1907802	DNS	FESS	35	1	1	0	0	0	Nausea and vomiting	air	ASA PS I I
ameena	32f	1876639	csom	myringoplasty	40	1	1	0	0	0	Nausea and vomiting	air	ASA PS I
sandhiya	17f	1655341	CSOM	cortical mastoidectomy	35	1	1	0	0	0	Nausea and vomiting	air	ASA PS I